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USPT,JPAB,EPAB,DWPI	chicheportiche-y\$.in.	1	<u>L3</u>
USPT,JPAB,EPAB,DWPI	chercheportiche-y\$.in.	0	<u>L2</u>
USPT,JPAB,EPAB,DWPI	browning-j\$.in.	329	<u>L1</u>

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USPT,JPAB,EPAB,DWPI	117 and trell	0	<u>L18</u>
USPT,JPAB,EPAB,DWPI	116 and related	779	<u>L17</u>
USPT,JPAB,EPAB,DWPI	115 and family	847	<u>L16</u>
USPT,JPAB,EPAB,DWPI	114 and (necrosis adj1 factor)	1658	<u>L15</u>
USPT,JPAB,EPAB,DWPI	19 or 110 or 111 or 112 or 113	16573	<u>L14</u>
USPT,JPAB,EPAB,DWPI	((536/23.1 536/23.5 536/25.1)!.CCLS.)	6189	<u>L13</u>
USPT,JPAB,EPAB,DWPI	((424/93.2 424/93.21 424/93.7)!.CCLS.)	868	<u>L12</u>
USPT,JPAB,EPAB,DWPI	((530/350 530/351)!.CCLS.)	5710	<u>L11</u>
USPT,JPAB,EPAB,DWPI	((514/44 514/885)!.CCLS.)	1817	<u>L10</u>
USPT,JPAB,EPAB,DWPI	((435/69.1 435/70.1 435/70.3 435/455 435/325)!.CCLS.)	7869	<u>L9</u>
USPT,JPAB,EPAB,DWPI	trell and (peptide or polypeptide or protein)	4	<u>L8</u>
USPT,JPAB,EPAB,DWPI	trell and (tumor or tumour)	2	<u>L7</u>
USPT,JPAB,EPAB,DWPI	trell	83	<u>L6</u>
USPT,JPAB,EPAB,DWPI	11 and trell	1	<u>L5</u>
USPT,JPAB,EPAB,DWPI	chichportiche-y\$.in.	0	<u>L4</u>
USPT,JPAB,EPAB,DWPI	chicheportiche-y\$.in.	1	<u>L3</u>
USPT,JPAB,EPAB,DWPI	chercheportiche-y\$.in.	0	<u>L2</u>
USPT,JPAB,EPAB,DWPI	browning-j\$.in.	329	<u>L1</u>

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=> s review/dt

L1 1390059 REVIEW/DT

=> s l1 and (tumor or tumour)(w)(necrosis factor)/ab,bi

201981 TUMOR
449 TUMOR
44818 NECROSIS/AB
475068 FACTOR/AB
26300 (NECROSIS FACTOR)/AB
(NECROSIS(W)/FACTOR)/AB
55889 NECROSIS/BI
569092 FACTOR/BI

34632 (NECROSIS FACTOR)/BI
(NECROSIS(W)/FACTOR)/BI
34620 (TUMOR OR TUMOUR)(W)(NECROSIS FACTOR)/AB,BI
L2 2613 L1 AND (TUMOR OR TUMOUR)(W)(NECROSIS FACTOR)/AB,BI

=> s l2 and related/ab,bi

574704 RELATED/AB
656648 RELATED/BI
L3 206 L2 AND RELATED/AB,BI

=> s l3 and family/ab,bi

97856 FAMILY/AB
107175 FAMILY/BI
L4 42 L3 AND FAMILY/AB,BI

=> s l4 and function/ab,bi

900638 FUNCTION/AB
1053643 FUNCTION/BI
L5 16 L4 AND FUNCTION/AB,BI

=> s l5 and homolog/ab,bi

683 HOMOLOG/AB
5594 HOMOLOG/BI
L6 0 L5 AND HOMOLOG/AB,BI

=> d l5 1- bit ab

YOU HAVE REQUESTED DATA FROM 16 ANSWERS -
CONTINUE? Y/(N)/Y

L5 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:763123 CAPLUS
DN 132:44343
TI Apoptosis regulating proteins as targets of therapy for hematological malignancies

AU Kombau, Steven M.; Komopleva, Marina; Andreotti, Michael
CS Department of Blood and Marrow Transplantation, Section of Molecular Hematology and Therapy, The University of Texas M. D. Anderson Cancer Center
SO Expert Opin. Invest. Drugs (1999), 8(12), 2027-2057
CODEN: EOIDER; ISSN: 1354-3784
PB Ashley Publications
DT Journal; ***General Review***
LA English
AB A review with 306 refs. Most chemotherapeutic agents used in the treatment of haematol. malignancies cause cell death by inducing

apoptosis through undefined means. The discovery of the proteins involved in apoptosis and the description of apoptotic pathways suggest new potential targets for therapeutic intervention. Both "intrinsic" and "extrinsic" pathways can be activated sep., but activation of caspases appears central to most apoptotic pathways. Novel approaches attempt to induce apoptosis by directly targeting a portion of an apoptotic pathway. Agents that trigger signalling of Fas or ***tumour*** ***necrosis***

factor (TNF) ***related*** apoptosis inducing ligand (TRAIL) receptor seek to induce the extrinsic pathway at the cell surface.

The BCL-2 ***family*** of proteins seems central to the regulation of these apoptotic pathways that involve mitochondrial sequestration or the release of cytochrome c, with subsequent activation of Apaf-1, caspase-9 and caspase-3. The activity of this ***family*** may depend upon both the phosphorylation state of different members and the relative level of pro- and anti-apoptotic members. New agents such as the staurosporine analog UCN-01 and bryostatin are thought to affect apoptosis induction by altering BCL-2 phosphorylation. Others, such as BCL-2 antisense and ATRA attempt to modulate the protein levels to promote apoptosis. Direct activation of caspase-3 is a probable target, but as yet no agent with this direct ***function*** is in trial. Clin. trials of several agents have been completed or are underway. It is likely that target particular points in apoptosis pathways will have antileukemia/lymphoma activity, however, the optimal utilization may involve combination with other more conventional agents that also activate apoptosis.

RE CNT 307

(1) Adida, C. Am J Pathol 1998, V152, P43 CAPLUS
(3) Akiyama, T. Anticancer Res 1999, V10, P67 CAPLUS
(4) Akiyama, T. Cancer Res 1997, V57, P1493 CAPLUS
(5) Altieri, E. Proc Natl Acad Sci USA 1992, V89, P7295 CAPLUS
(6) Altieri, D. FASEB J 1995, V9, P860 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999:744668 CAPLUS
DN 132:235500
TI ***Tumour*** ***necrosis*** ***factor*** receptors in systemic

inflammation
 AU Lin, E.; Calvano, S. E.; Lowry, S. F.
 CS Department of Surgery, New York Hospital, Queens Flushing,
 NY, USA
 SO Update Intensive Care Emerg Med (2000), 31(Immune
 Response in the
 Critically Ill), 365-384
 CODEN: JUCMKN, ISSN: 0933-6788
 PB Springer-Verlag
 DT Journal: ***General Review***
 LA English
 AB A review with 114 refs. of what is known about ***tumor***
 necrosis ***factor*** (TNF) receptor
 function and
 signal transduction as they relate to inflammation. Due to
 similarities
 in receptor structure and signaling pathways, the ***function***
 of
 related TNF receptor ***family*** members during
 systemic
 inflammation is also highlighted. Finally, the authors focus on
 clin.-derived data, beginning with immunocyte TNF receptor
 alterations
 exhibited during acute systemic inflammation and culminating in
 potential
 clin. implications stemming from such changes.
 RE CNT 114
 RE
 (1) Abraham, E. JAMA 1997, V277, P1331 CAPLUS
 (2) Adam, D. Biochem J 1998, V333, P343 CAPLUS
 (3) Adenka, D. J Exp Med 1992, V175, P323 CAPLUS
 (4) Arraraja, J. J Clin Invest 1997, V99, P763 CAPLUS
 (8) Bazzoni, F. N Engl J Med 1996, V334, P1717 CAPLUS
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L5 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1999-664951 CAPLUS
 DN 132:11436
 TI Interleukin-18
 AU Dinarello, Charles A.
 CS Department of Medicine, Division of Infectious Diseases, B168,
 University
 of Colorado Health Sciences Center, Denver, CO, 80262, USA
 SO Methods (Orlando, Fla.) (1999), 19(1), 121-132
 CODEN: MATHDE, ISSN: 1046-2023
 PB Academic Press
 DT Journal: ***General Review***
 LA English
 AB A review with 81 refs. summarizing the present knowledge on
 IL-18, to give
 an insight into the future perspectives for its possible use as vaccine
 adjuvant. Formerly called interferon (IFN) gamma, inducing factor
 (GIF), IL-18 is the new name of a novel cytokine that plays an
 important
 role in the T-helper type 1 (Th1) response, primarily by its ability
 to
 induce IFN gamma, prodrn. in T cells and natural killer (NK) cells.

not here reduced 7/12

IL-18
 is ***related*** to the IL-1 ***family*** in terms of
 structure. ***family*** and ***function***. Also similar to
 IL-1 beta, IL-18 is synthesized as a biol. inactive precursor mol.
 lacking a signal peptide which requires cleavage into an active,
 mature
 mol. by the intracellular cysteine protease called
 IL-1 beta-converting
 enzyme (ICE, also called caspase-1). The activity of mature IL-18
 is
 closely ***related*** to that of IL-1. IL-18 induces gene
 expression
 and synthesis of ***tumor*** ***necrosis*** ***factor***
 (TNF), IL-1, Fas ligand, and several chemokines. The activity of
 IL-18 is
 via an IL-18 receptor (IL-18R) complex. This IL-18R complex is
 made up of
 a binding chain termed IL-18R alpha, a member of the IL-1
 receptor
 family previously identified as the IL-1 receptor-
 protein (IL-1R), and a signaling chain, also a member of the
 IL-1R
 family. The IL-18R complex recruits the
 IL-1R-activating kinase
 (IRAK) and TNFR-associated factor-6 (TRAF-6) which
 phosphorylates nuclear
 factor kappa B (NF kappa B)-inducing kinase (NIK) with
 subsequent
 activation of NF kappa B. Thus, on the basis of primary structure,
 3-dimensional structure, receptor ***family***, signal
 transduction
 pathways, and biol. effects, IL-18 appears to be a new member of
 the IL-1
 family. (c) 1999 Academic Press.
 RE CNT 81
 RE
 (1) Adachi, O. Immunity 1998, V9, P143 CAPLUS
 (2) Barbulescu, K. J Immunol 1998, V160, P3642 CAPLUS
 (3) Bohm, E. J Immunol 1998, V160, P299 CAPLUS
 (4) Borsesh, D. Eur Cytokine New 1998, V9, P205 CAPLUS
 (5) Born, T. Biol Chem 1998, V273, P2945 CAPLUS
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L5 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1999-6641810 CAPLUS
 DN 131:349988
 TI The ***tumor*** ***necrosis*** ***factor*** (TNF)
 family and ***related*** molecules
 AU Wallach, David, Bigda, Jack, Engelmann, Hartmut
 CS Department of Biological Chemistry, The Weizmann Institute of
 Science,
 Rehovot, 76100, Israel
 SO Cytokine Network Immune Funct. (1999), 51-84, Editor(s):
 Theze, Jacques.

Publisher: Oxford University Press, Oxford, UK.
 CODEN: 68GGAA
 DT Conference: ***General Review***
 LA English
 AB A review with 31 refs. Topics discussed include common
 features of
 family members, occurrence of ligands and receptors,
 common and
 distinct effects of the TNF ligand and receptor families, cellular
 origins
 of TNFs and their receptors, ***functions*** of TNFs,
 structure-
 function relationships in TNFs and their receptors,
 intracellular
 domains of TNF receptors, HVEM and LIGHT, CD95, Apo-3 and
 Apo3L, TRAIL,
 CAR1, Osteoprotegerin, TRANCE, RANK, CD40, CD40L,
 GITR, OX40, TACI, and
 APRIL.
 RE CNT 10
 RE
 (1) Beutler, B. Science 1994, V264, P667 CAPLUS
 (2) Cosman, D. Stem Cells 1994, V12, P440 CAPLUS
 (4) Gruss, H. Blood 1995, V85, P3378 CAPLUS
 (5) Meakin, S. Trends Neurosci 1992, V15, P323 CAPLUS
 (7) Smith, C. Cell 1994, V76, P939 CAPLUS
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L5 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1999-636341 CAPLUS
 DN 131:226786
 TI Aberrant apoptotic signals in tumorigenesis
 AU Sakemura, Daiichu
 CS Walter Cancer Inst. Dep. Med. Chem. Mol. Pharmacol., Purdue
 Univ. Cancer
 Cent., USA
 SO Jikken Igaku (1999), 17(14), 1911-1918
 CODEN: JIGGEF, ISSN: 0288-5514
 PB Yodoshu
 DT Journal: ***General Review***
 LA Japanese
 AB A review with 41 refs. on (1) p53 activation and apoptosis
 induced by DNA
 damage, (2) p53-mediated apoptosis induced by oxidative stress,
 (3) roles
 of p19ARF, BIN1, Fas/Fas ligand, and cytochrome c in
 c-myc-dependent
 apoptosis and tumorigenesis, (4) structure and pathophysiol.
 functions of TNF receptor ***family*** mols. in
 apoptosis, and
 (5) possible use of TRAIL (TNF: ***related***
 apoptosis-inducing
 ligand) in cancer treatment.
 L5 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1999-404130 CAPLUS
 DN 131:183537

2095.568

- TI TRANCE is a TNF ***family*** member that regulates dendritic cell and osteoclast ***function***
- AU Wong, Brian R.; Jostein, Regis; Choi, Yongwon
CS Laboratory of Immunology, The Rockefeller University, New York, NY, 10021, USA
- SO J. Leukocyte Biol. (1999) 65(6), 715-724
CODEN: JLBIET; ISSN: 0741-5400
PB Federation of American Societies for Experimental Biology
DT Journal; ***General Review***
LA English
AB A review with 75 refs. ***Tumor*** ***necrosis*** ***factor***
related activation-induced cytokine (TRANCE) (TNF). ***related*** emerging as a key regulator of the immune system and of bone development and homeostasis. TRANCE is expressed on activated T cells and activates mature dendritic cells (DC), suggesting that it plays a role in the T cell-DC interaction during an immune response. Furthermore, TRANCE is expressed on osteoblasts stimulated with vitamin D3, dexamethasone, and parathyroid hormone. TRANCE, when expressed on osteoblasts, induces osteoclastogenesis and osteoclast activation, suggesting that it links known calcitropic hormones to bone resorption. TRANCE mediates its effects via the TRANCE-receptor (TRANCE-R/RANK), whereas its activity can be inhibited by the sol. decoy receptor osteoprotegerin/osteoclast inhibitory factor (OPG/OCIF). OPG can be neutralized by another TNF- ***family*** member, the TNF- ***related*** apoptosis-inducing ligand (TRAIL), suggesting that TRANCE is part of a complex cytokine network that regulates a diverse set of ***functions***. The authors discuss the current literature describing TRANCE and its receptors and its role in controlling DC and osteoclast ***function***.
- RE CNT 75
- RE (1) Anderson, D.; Nature 1997, V390, P175 CAPLUS
(2) Beuterie, P.; Science 1988, V242, P540 CAPLUS
(3) Banchereau, J.; Nature 1998, V392, P245 CAPLUS
(4) Bennett, S.; Nature 1998, V393, P478 CAPLUS
(5) Buery, N.; Genes Dev 1998, V12, P1260 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L5 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999-315231 CAPLUS
- DN 131:10093
TI To die or not to die-the quest of the TRAIL receptors
AU Degli-Esposti, Mariapia
CS Department of Microbiology, QELI Medical Centre, The University of Western Australia, Perth, 6009, Australia
SO J. Leukocyte Biol. (1999) 65(5), 535-542
CODEN: JLBIET; ISSN: 0741-5400
PB Federation of American Societies for Experimental Biology
DT Journal; ***General Review***
LA English
AB A review with 59 refs. The last 18 mo have witnessed the characterization of several new members of the ***tumor*** ***necrosis*** ***factor*** (TNF) receptor ***family***. Among these are five receptors for the cytotoxic ligand TRAIL (TNF- ***related*** apoptosis-inducing ligand). Two of these receptors, TRAIL-R1 and TRAIL-R2, contain classical cytoplasmic death domains and are able to transduce an apoptotic signal. The others lack functional death domains and are not able to promote cell death. Indeed, one of the receptors for TRAIL, osteoprotegerin (OPG) is a sol. protein whose activities so far have been shown to be inhibition of osteoclastogenesis and increased bone d. in vivo. The existence of multiple receptors for TRAIL suggests an unexpected complexity to TRAIL-mediated biol. ***functions***.
- RE CNT 59
- RE (1) Amakawa, R.; Cell 1996, V84, P551 CAPLUS
(2) Anderson, D.; Nature 1997, V390, P175 CAPLUS
(3) Bodmer, J.; Immunity 1997, V6, P79 CAPLUS
(6) Browning, J.; J Exp Med 1996, V183, P867 CAPLUS
(8) Chidiebere, Y.; J Biol Chem 1997, V272, P32401 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L5 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999-182258 CAPLUS
DN 131:27970
TI A New Member of ***Tumor*** ***Necrosis*** ***Factor*** Ligand ***Family***, ODF/OPG/TRANCE/RANKL, Regulates Osteoclast Differentiation and ***Function***
- AU Takahashi, Neoyuki; Udegawa, Nobuyuki; Suda, Tetsuo
CS Department of Biochemistry, School of Dentistry, Shiga University, Tokyo, 142-8555, Japan
SO Biochem. Biophys. Res. Commun. (1999), 256(3), 449-455
CODEN: BBRCAG; ISSN: 0006-291X
PB Academic Press
DT Journal; ***General Review***
- LA English
AB A review and discussion with 55 refs. Osteoclasts, the multinucleated giant cells that resorb bone, develop from monocyte-macrophage lineage cells. Osteoblasts or bone marrow stromal cells have been suggested to be involved in osteoclastic bone resorption. The recent discovery of new members of the ***tumor*** ***necrosis*** ***factor*** (TNF) receptor-ligand ***family*** has elucidated the precise mechanism by which osteoblasts/stromal cells regulate osteoclast differentiation and ***function***. Osteoblasts/stromal cells express a new member of the TNF-ligand ***family*** osteoclast differentiation factor (ODF)/osteoprotegerin ligand (OPG)/TNF- ***related*** activation-induced cytokine (TRANCE)/receptor activator of NF-kB ligand (RANKL)* as a membrane associated factor. Osteoclast precursors which possess RANK, a TNF receptor ***family*** member, recognize ODF/OPG/TRANCE/RANKL through cell-to-cell interaction with osteoblasts/stromal cells, and differentiate into osteoclasts in the presence of macrophage colony-stimulating factor. Mature osteoclasts also express RANK, and their bone-resorbing activity is also induced by ODF/OPG/TRANCE/RANKL which osteoblasts/stromal cells possess. Osteoprotegerin (OPG)/osteoclastogenesis inhibitory factor (OCIF)/TNF receptor-like mol. 1 (TRL1) is a sol. decoy receptor for ODF/OPG/TRANCE/RANKL. Activation of NF-kB and c-Jun N-terminal kinase through the RANK-mediated signaling system appears to be involved in differentiation and activation of osteoclasts. (c) 1999 Academic Press.
- RE CNT 55
- RE (1) Aleva, T.; Bone 1998, V2, P495 CAPLUS
(2) Anderson, D.; Nature 1997, V390, P175 CAPLUS
(5) Brandstrom, H.; Biochem Biophys Res Commun 1998, V248, P454 CAPLUS
(6) Buery, N.; Genes Dev 1998, V12, P1260 CAPLUS
(7) Chambers, T.; Proc Natl Acad Sci USA 1993, V90, P5578 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L5 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1999-156605 CAPLUS
DN 130:178865
TI Structural biology of apoptosis proteins. Recent advances in structural

analysis of TNF. ***related***, Fas- ***related***, Bcl-2
 family and caspase ***family*** proteins
 AU Arizumi, Masaharu, Ohia, Shingo
 CS Dep. Struct. Biol., Biomol. Eng. Res. Inst., Suita, 565-0874,
 Japan
 SO Tempakushitsu Kakusan Koso (1999), 44(4), 395-403
 CODEN: TAKKAJ, ISSN: 0039-9450
 PB Kyoritsu Shuppan
 DT Journal, ***General Review***
 LA Japanese
 AB A review with 25 refs., on (1) transduction of apoptotic signals in
 Caenorhabditis elegans, (2) TNF- or Fas ligand-mediated signal
 transduction in mammals, (3) mitochondria-mediated signal
 transduction of
 apoptosis, (4) conformation of TNF and its receptor, (5)
 three-dimensional
 structure of Fas death domain and Fas-assocd. protein sith death
 domain
 (FADD), (6) structure and ***function*** of Bcl-2
 family
 proteins, an d(7) crystal structure of caspase ***family***
 proteins.
 L5 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1998;761671 CAPLUS
 DN 130:134227
 TI Control of neuronal survival by neurotrophins
 AU Fried, Jose Marie; Casademunt, Elisabeth; Dechant, Georg;
 Barde,
 Yves-Alain
 CS Max Planck Inst. Psychiatry, Planegg-Martinsried, Germany
 SO Veth. - K. Ned. Akad. Wet., Afr. Naturkd., Tweede Rocks
 (1998),
 100(Pharmaceutical Intervention in Apoptotic Pathways), 87-96
 CODEN: VNAWAG, ISSN: 0373-465X
 PB North-Holland
 DT Journal, ***General Review***
 LA English
 AB A review with 59 refs. Neurotrophins are ***related***
 secretory
 proteins that control cell survival in the nervous system. All can
 prevent programmed cell death by binding to specific cell surface
 receptors belonging to a ***family*** of tyrosine kinase
 receptors.
 As these receptors are expressed in subgroups of developing
 neurons,
 interference with the ***function*** of these receptors or of
 their
 ligands leads to selective neuronal deficits in the nervous system.
 All
 neurotrophins also bind to another receptor designated the
 neurotrophin
 receptor p75. This member of the ***family***
 necrosis
 factor receptor ***family*** can be activated by
 nerve growth
 factor, leading to the death of neurons in the developing nervous

system.
 Thus, the neurotrophin nerve growth factor controls cell nos. in
 opposite
 ways by its ability to activate 2 different receptors.
 RE CNT 60
 RE
 (1) Bardeid, M.; J Neurobiol 1994, V25, P1386 CAPLUS
 (2) Behrwell, M.; Annu Rev Neurosci 1995, V18, P223 CAPLUS
 (3) Bovolenta, P.; J Neuroscience 1996, V16, P4402 CAPLUS
 (4) Carter, B.; Science 1996, V272, P542 CAPLUS
 (5) Casaccia-Bonelli, P.; Nature 1996, V383, P716 CAPLUS
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L5 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1998;375207 CAPLUS
 DN 129:11786
 TI Neurotrophins: the biological paradox of survival factors eliciting
 apoptosis
 AU Casaccia-Bonelli, Patricia; Kong, Haoyoung; Chao, Moses V.
 CS Molecular Neurobiology Program, Skirball Institute, NY, 10016,
 USA
 SO Cell Death Differ. (1998), 5(5), 357-364
 CODEN: CDDIEK, ISSN: 1350-9047
 PB Stoddan Press
 DT Journal, ***General Review***
 LA English
 AB A review with approx. 50 refs. Neurotrophins are target-derived
 sol.
 polypeptides required for neuronal survival. Binding of
 neurotrophins to
 Trk receptor tyrosine kinases initiate signaling cascades that
 promote
 cell survival and differentiation. All ***family*** members
 bind to
 another receptor (p75NTR), which belongs to the ***family***
 necrosis ***factor*** superfamily. Hence, nerve
 growth factor
 (NGF) and ***related*** trophic factors are unique in that two
 sep.
 receptor types are utilized. Although the biol. ***function***
 of
 p75NTR has been elusive, it has been suggested to mediate
 apoptosis of
 developing neurons in the absence of Trk receptors. This presents
 a
 tantalizing paradigm, in which life-death decisions of cells are
 dependent
 upon the expression and action of two different receptors with
 distinctive
 signaling mechanisms. In the presence of TrkA receptors, p75 can
 participate in the formation of high affinity binding sites and
 enhanced
 NGF responsiveness leading to a survival signal. In the absence of
 TrkA
 receptors, p75 can generate, in only specific cell populations, a
 death
 signal. Here we discuss the unique features and implications of this

unusual signal transduction system.
 L5 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1998;292210 CAPLUS
 DN 129:94051
 TI The TRAIL of death
 AU Goodwin, R. G.; Smith, C. A.
 CS Immux Corporation, Seattle, WA, 98101, USA
 SO Apoptosis (1998), 3(2), 83-88
 CODEN: APOPFN, ISSN: 1360-8185
 PB Rapid Science Ltd
 DT Journal, ***General Review***
 LA English
 AB A review with 44 refs. The TNF ligand ***family***
 member termed
 TRAIL has been shown to induce apoptosis in a wide variety of
 transformed
 cell lines. The normal ***functions*** of this cytokine in vivo
 system
 remain, however, relatively unknown. The complexity of this biol.
 system
 has now increased unexpectedly with the identification of four
 distinct
 receptors for TRAIL, two of which have cytoplasmic death
 domains. This
 review will describe the known biol. effects of TRAIL, as well as
 the
 structure and possible ***functions*** of its recently identified
 receptors.
 L5 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2000 ACS
 AN 1997;749108 CAPLUS
 DN 128:43897
 TI Eph ***family*** receptors and ligands in vascular cell
 targeting and
 assembly
 AU Stein, Elke; Schoecklmann, Harald; Daniel, Thomas O.
 CS Department of Pharmacology, Vanderbilt University Medical
 Center,
 Nashville, TN, USA
 SO Trends Cardiovasc. Med. (1997), 7(6), 329-334
 CODEN: TCMDEQ, ISSN: 1050-1738
 PB Elsevier
 DT Journal, ***General Review***
 LA English
 AB A review, with 52 refs. Members of the Eph ***family*** of
 receptor
 tyrosine kinases det. neural cell aggregation and targeting behavior,
 functions that are also crit. in vascular assembly and
 remodeling.
 Among this class of diverse receptors EphA2 (Eck) and EphB1
 (ELK)
 represent prototypes for two receptor subfamilies distinguished by
 high-affinity interaction with either glycoprophosphatidylinositol
 (GPI)-linked or transmembrane ligands, resp. EphA2 participates in
 angiogenic responses to ***family*** ***necrosis***
 factor
 (TNF) through an autocrine loop affecting endothelial cell

not here
 reduced also

migration EphB1 and its ligand Epruin-B1 (LERK-2) are important determinants of assembly of endothelial cells from the microvasculature of the kidney where both are expressed in endothelial progenitors and in glomerular microvascular endothelial cells. Ephrin-B1 activation of EphB1 promotes assembly of these cells into capillary-like structures. Interaction trap approaches have identified downstream signaling proteins that complex with ligand-activated EphA2 or EphB1, including nonreceptor tyrosine kinase and SH2 domain-contg. adapter proteins. The Grb 10 adapter is one of a subset that binds activated EphB1, but not EphA2, defining distinct signaling mechanisms for these ***related*** endothelial receptors. On the basis of observations in vascular endothelial cells and recent results defining Eph receptor and ligand roles in neural cell targeting, we propose that these receptors direct cell-cell recognition events that are crit. in vasculogenesis and angiogenesis.

L5 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1996:624415 CAPLUS
DN 125:272074
TI Common aspects of the signal transduction mechanism of the Epstein-Barr virus (EBV) transforming protein latent membrane protein LMP1 and members of TNF receptor ***family***
AU Hareida, Shizuko; Mostoslav, George
CS Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, USA
SO Saibo Kogaku (1996), 15(9), 1241-1248
CODEN: SAKOEO; ISSN: 0287-3796
DT Journal; ***General Review***
LA Japanese
AB A review with 32 refs., on LMP1 and malignant tumor, structure and ***function*** of LMP1, recombinant EBV expts., investigation of LMP1 binding protein, structure and ***function*** of TNF receptor assoc. factor (TRAF) and LMP1 and TRAF. ***related*** transformation model.

L5 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1997:529436 CAPLUS
DN 117:129436
TI Interleukin-8, a chemotactic and inflammatory cytokine
AU Baggiolini, Marco; Clark-Lewis, Ian
CS Theodor-Kocher Inst., Univ. Bern, Bern, CH-3000, Switz

SO FEBS Lett. (1992), 307(1), 97-101
CODEN: FEBLAL; ISSN: 0014-5793
DT Journal; ***General Review***
LA English
AB A review with 38 refs. Interleukin-8 (IL-8) belongs to a ***family*** of small, structurally ***related*** cytokines similar to platelet factor 4. It is produced by phagocytes and mesenchymal cells exposed to inflammatory stimuli (e.g., interleukin-1 or ***tumor*** inducing ***necrosis*** **factor***) and activates neutrophils eliciting chemotaxis, exocytosis and the respiratory burst. In vivo, IL-8 massive neutrophil accumulation at the site of injection. Five neutrophil-activating cytokines similar to IL-8 in structure and ***function*** have been identified recently. IL-8 and the ***related*** cytokines are produced in several tissues upon infection, inflammation, ischemia, trauma etc., and are thought to be the main cause of local neutrophil accumulation.

L5 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1991:623497 CAPLUS
DN 115:223497
TI A new superfamily of cell surface proteins ***related*** to the nerve growth factor receptor
AU Mallett, Susan; Barclay, A. Neil
CS Sir William Dunn Sch. Pathol., Univ. Oxford, Oxford, UK
SO Immunol. Today (1991), 12(7), 220-3
CODEN: IMTOD8; ISSN: 0167-4619
DT Journal; ***General Review***
LA English
AB A review, with 33 refs., of the mol. functional features of the proteins. These include 2 lymphocyte proteins of unknown ***function*** and 2 receptors for ***tumor*** ***necrosis*** ***factor***. These are cysteine-rich membrane proteins and probably ***function*** as receptors for cytokines.

=> d his

(FILE HOME ENTERED AT 11:21:22 ON 10 JUL 2000)

FILE CAPLUS ENTERED AT 11:21:27 ON 10 JUL 2000

L1 1390059 S REVIEW/DT
L2 2613 S LI AND (TUMOR OR TUMOUR)XVXNECROSIS
FACTOR/AB,BI
L3 206 S L2 AND RELATED/AB,BI
L4 42 S L3 AND FAMILL Y/AB,BI
L5 16 S L4 AND FUNCTION#/AB,BI

L6 0 S L5 AND HOMOLOGY/AB,BI
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COST IN U.S. DOLLARS	SINCE FILE	TOTAL
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SINCE FILE TOTAL	ENTRY SESSION	-8.90 -8.90
CA SUBSCRIBER PRICE		
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Adonis

L4 ANSWER 5 OF 11 MEDLINE
AN 1999175482 MEDLINE
DN 99175482
TI Identification of a new member of the **tumor necrosis factor family** and its receptor, a human ortholog of mouse GITR.
AU Gurney A L; Marsters S A; Huang R M; Pitti R M; Mark D T; Baldwin D T; Gray A M; Dowd A D; Brush A D; Heldens A D; Schow A D; Goddard A D; Wood W
I; Baker K P; Godowski P J; Ashkenazi A
CS Department of Molecular Biology Genentech Inc. 1 DNA Way South San Francisco California 94080 USA.
SO CURRENT BIOLOGY, (1999 Feb 25) 9 (4) 215-8.
Journal code: B44. ISSN: 0960-9822.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199906
EW 19990604
AB The tumor necrosis factor (TNF) and TNF receptor (TNFR) gene superfamilies regulate diverse biological functions, including cell proliferation, differentiation, and survival [1] [2] [3]. We have identified a new TNF-**related** ligand, designated human GITR ligand (hGITRL), and its human receptor (hGITR), an ortholog of the recently discovered murine glucocorticoid-induced TNFR-**related** (mGITR) protein [4]. The hGITRL gene mapped to chromosome 1q23, near the gene for the TNF homolog Fas/CD95 ligand [5]. The hGITR gene mapped to chromosome 1p36, near a cluster of five genes encoding TNFR homologs [1] [6]. We found hGITRL mRNA in several peripheral tissues, and detected hGITRL protein on cultured vascular endothelial cells. The levels of hGITR mRNA in tissues were generally low; in peripheral blood T cells, however, antigen-receptor stimulation led to a substantial induction of hGITR transcripts. Cotransfection of hGITRL and hGITR in embryonic kidney 293 cells activated the anti-apoptotic transcription factor NF-kappaB, via a pathway that appeared to involve TNFR-associated factor 2 (TRAF2) [7] and NF-kappaB-inducing kinase (NIK) [8]. Cotransfection of hGITRL and hGITR in Jurkat T leukemia cells inhibited antigen-receptor-induced cell death. Thus, hGITRL and hGITR may modulate T lymphocyte survival in peripheral tissues.

*****STN Columbus*****

FILE HOME/ ENTERED AT 10:33:47 ON 10 JUL 2000

=> file medline

COST IN U.S. DOLLARS	ENTRY	SINCE FILE	TOTAL
FULL ESTIMATED COST	0.15	0.15	

FILE MEDLINE/ ENTERED AT 10:33:58 ON 10 JUL 2000

FILE LAST UPDATED: 6 JUL 2000 (20000706/UP). FILE COVERS 1960 TO DATE.

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OLDMEDLINE, data from 1960 through 1965 from the Cumulated Index Medicus (CINA), has been added to MEDLINE. See HELP CONTENT for details.

Left, right, and simultaneous left and right truncation are available in the Basic Index. See HELP SIELDS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

=> s trill/ab,bi

1 TREL/BI
5348796 AB/FA
1 TREL/AB
(TREL/BI (L) AB/FA)
1 TREL/BI
1 TREL/AB,BI

=> d bib ab

L1 ANSWER 1 OF 1 MEDLINE
AN 76058643 MEDLINE
DN 76058643
TI Hydantoin derivatives and malignancies of the haemopoietic system
AU Bichel J
SO ACTA MEDICA SCANDINAVICA, (1975 Oct) 198 (4) 327-8.
Journal code: 14G. ISSN: 0001-6101.
CY Sweden

DT Journal, Article: (JOURNAL ARTICLE)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 197603

AB Two patients are described who developed malignant lymphoma (lymphosarcoma) after diphenylhydantoin therapy because of epilepsy.

Malignant lymphoma in a few patients receiving this medication has been described earlier. The literature has been reviewed and discussed recently by Rausing and ***Trell*** (2).

=> s tumor necrosis factor family related protein#/ab,bi

402104 TUMOR/BI
106882 NECROSIS/BI
444330 FACTOR/BI
244109 FAMIL Y/BI
561805 RELATED/BI
1183373 PROTEIN#/BI
5348796 AB/FA
0 TUMOR NECROSIS FACTOR FAMIL Y RELATED
PROTEIN#/AB

((TUMOR(W)NECROSIS(W)FACTOR(W)FAMIL Y(W)RELATE
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106882 NECROSIS/BI
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0 TUMOR NECROSIS FACTOR FAMIL Y RELATED
PROTEIN#/BI

((TUMOR(W)NECROSIS(W)FACTOR(W)FAMIL Y(W)RELATE
D(W)PROTEIN#)Y/BI
L2 0 TUMOR NECROSIS FACTOR FAMIL Y RELATED
PROTEIN#/AB,BI

=> s tumor necrosis factor family/ab,bi

402104 TUMOR/BI
106882 NECROSIS/BI
444330 FACTOR/BI
244109 FAMIL Y/BI
5348796 AB/FA
40 TUMOR NECROSIS FACTOR FAMIL Y/AB
(TUMOR(W)NECROSIS(W)FACTOR(W)FAMIL Y)BI
(L) AB/FA)
402104 TUMOR/BI
106882 NECROSIS/BI
444330 FACTOR/BI
244109 FAMIL Y/BI

53 TUMOR NECROSIS FACTOR FAMIL Y/BI

((TUMOR(W)NECROSIS(W)FACTOR(W)FAMIL Y)BI
L3 53 TUMOR NECROSIS FACTOR FAMIL Y/AB,BI

=> s l3 and related/ab,bi

561805 RELATED/BI
5348796 AB/FA
454744 RELATED/AB
(RELATED/BI (L) AB/FA)
561805 RELATED/BI
L4 11 L3 AND RELATED/AB,BI

=> d l - bib ab

YOU HAVE REQUESTED DATA FROM 11 ANSWERS -
CONTINUE? Y/(N)Y

L4 ANSWER 1 OF 11 MEDLINE
AN 2000219071 MEDLINE
DN 20219071

TI Prognostic relevance of altered Fas (CD95)-system in human breast cancer.

AU Motolaise M, Buglioni S, Baccalenti C, Cardarelli M A, Giannarelli D, Botti C, Natali P G, Conzetti A, Venzani F M

CS Pathology Department, Regina Elena Cancer Institute, Rome, Italy

SO INTERNATIONAL JOURNAL OF CANCER, (2000 Mar 20) 89 (2) 127-32.

Journal code: GQU. ISSN: 0020-7136.

CY United States

DT Journal, Article: (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

EM 200006

EW 20000605

AB The Fas ligand (Fasl) and its receptor Fas (APO-1 or CD95) are members, respectively, of the ***tumor*** ***necrosis*** ***factor***

****family**** that, upon interaction with each other, play a key role in the initiation of one apoptotic pathway. Faulty regulation of the Fas system has been described in a variety of human tumors with different histogenetic origin. Here, we describe the expression and distribution of Fas receptor and ligand pair antigens in surgical samples collected from a cohort of 186 patients bearing breast neoplasms (45 benign and 141 malignant lesions). Immunoperoxidase staining of formalin-fixed tissues showed that 91.1% of benign lesions expressed Fas, which was present in

only 36.7% of malignant tumors. On the other hand, FasL was found positive in 22.2% of benign neoplasms and up-regulated in situ as well as invasive carcinomas (53.9%). Moreover, in malignant tumors, the expression of receptor and ligand antigens appeared to be inversely ***related***

. When these findings were correlated with pathological parameters of prognostic relevance, a significant association was observed between FasL and the presence of metastatic lymph nodes and larger tumor size while Fas expression correlated to node-negative status and smaller tumor size. Patients with Fas positive tumors exhibited longer disease-free survival than those with Fas-negative carcinoma while FasL did not influence patient outcome. These relationships indicate that benign and malignant mammary lesions are characterized by differential cellular expression of Fas and FasL and suggest that a neoplastic Fas negative/FasL positive phenotype may be linked to breast cancer progression. Copyright 2000 Wiley-Liss, Inc.

L4 ANSWER 2 OF 11 MEDLINE
AN 2000130293 MEDLINE
DN 20130293
TI TRANCE, a ***tumor*** ***necrosis*** ***factor***
family member, enhances the longevity and adjuvant properties of dendritic cells in vivo.
AU Joesten R, Li H L, Ingulli E, Sarma S, Wang B R, Volopodskaja M, Steinman R
M, Choi Y
CS Laboratory of Cellular Physiology and Immunology, The Rockefeller University, New York, New York 10021, USA
NC A113013 (NIAD)
A144264 (NIAD)
DK39672 (NIDDK)
SO JOURNAL OF EXPERIMENTAL MEDICINE, (2000 Feb 7) 191 (3) 495-502.
Journal code: J2V. ISSN: 0022-1007.
CY United States
DT Journal, Article; (JOURNAL ARTICLE)
LA English
FS Cancer Journals, Priority Journals
EM 200006
AB Mature dendritic cells (DCs) are powerful antigen presenting cells that have the unique capacity to migrate to the T cell zone of draining lymph

nodes after subcutaneous injection. Here we report that treatment of antigen-pulsed mature DCs with tumor necrosis factor (TNF)-activation-induced cytokine (TRANCE), a TNF family member, before immunization enhances their adjuvant capacity and elicits improved T cell priming in vivo, such that both primary and memory T cell immune responses are enhanced. By enumerating migratory DCs in the draining lymph nodes and by studying their function in stimulating naive T cells, we show that one of the underlying mechanisms for enhanced T cell responses is an increase in the number of ex vivo antigen-pulsed DCs that are found in the T cell areas of lymph nodes. These results suggest that the longevity and abundance of mature DCs at the site of T cell priming influence the strength of the DC-initiated T cell immunity in situ. Our findings have the potential to improve DC-based immunotherapy; i.e., the active immunization of humans with autologous DCs that have been pulsed with clinically significant antigens ex vivo.

L4 ANSWER 3 OF 11 MEDLINE
AN 1999290669 MEDLINE
DN 99290669
TI Relation of TNF- ***related*** apoptosis-inducing ligand (TRAIL) receptor and FLICE-inhibitory protein expression to TRAIL-induced apoptosis of melanoma.
AU Zhang X D, Franco A, Myrnes K, Gray C, Nguyen T, Hershey P
CS Immunology and Oncology Unit, Department of Surgical Sciences, Newcastle, NSW, Australia
SO CANCER RESEARCH, (1999 Jun 1) 59 (11) 2747-53.
Journal code: CNF. ISSN: 0008-5472.
CY United States
DT Journal, Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals, Cancer Journals
EM 199909
EW 19990901
AB Past studies have shown that apoptosis mediated by TNF- ***related*** apoptosis-inducing ligand (TRAIL) is regulated by the expression of two death receptors [TRAIL receptor 1 (TRAIL-R1) and TRAIL-R2] and two decoy receptors [TRAIL-R3 and TRAIL-R4] that inhibit apoptosis. In previous studies, we have shown that TRAIL, but not other members of the ***tumor*** ***necrosis*** ***factor*** ***family*** induce

apoptosis in approximately two-thirds of melanoma cell lines. Here, we examined whether the expression of TRAIL-R at the mRNA and protein level in a panel of 28 melanoma cell lines and melanocytes correlated with their sensitivity to TRAIL-induced apoptosis. We report that at least three factors appear to underlie the variability in TRAIL-induced apoptosis. (a) Four of nine cell lines that were insensitive to TRAIL-induced apoptosis failed to express death receptors, and in two instances, lines were devoid of all TRAIL-Rs. Southern analysis suggested this was due to loss of the genes for the death receptors. (b) Despite the presence of mRNA for the TRAIL-R, some of the lines failed to express TRAIL-R protein on their surface. This was evident for TRAIL-R1 and more so for the TRAIL decoy receptors TRAIL-R3 and -R4. Studies on permeabilized cells revealed that the receptors were located within the cytoplasm and redistribution from the cytoplasm may represent a posttranslational control mechanism.

(c) Surface expression of TRAIL-R1 and -R2 (but not TRAIL-R3 and -R4) showed an overall correlation with TRAIL-induced apoptosis. However, certain melanoma cell lines and clones were relatively resistant to TRAIL-induced apoptosis despite the absence of decoy receptors and moderate levels of TRAIL-R1 and -R2 expression. This may indicate the presence of inhibitors within the cells, but resistance to apoptosis could not be correlated with expression of the caspase inhibitor FLICE-inhibitory protein mRNA for another TRAIL receptor, osteopontegin, was expressed in 22 of the melanoma lines but not on melanocytes. Its role in induction of apoptosis remains to be studied. These results appear to have important implications for future clinical studies on TRAIL.

L4 ANSWER 4 OF 11 MEDLINE
AN 1999207064 MEDLINE
DN 99207064
TI TRANCE, a ***tumor*** ***necrosis*** ***factor*** ***family*** member critical for CD40 ligand-independent T helper cell activation [see comments]

CM Comment in: J Exp Med 1999 Apr 5;189(7):1017-20
 AU Bachmann M F, Wong B R, Josien R, Steinman R M, Oxenius A, Choi Y
 CS Basel Institute for Immunology, CH 4005 Basel, Switzerland.
 NC GM40739 (NIGMS)
 AL44264 (NIAD)
 AL13013 (NIAD)
 +
 SO JOURNAL OF EXPERIMENTAL MEDICINE. (1999 Apr 5) 189 (7) 1025-31.
 Journal code: J2V. ISSN: 0022-1007.
 CY United States
 DT Journal, Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals; Cancer Journals
 EM 199907
 EW 19990702
 AB CD40 ligand (CD40L), a tumor necrosis factor (TNF) family member, plays a critical role in antigen-specific T cell responses in vivo. CD40L expressed on activated CD4(+) T cells stimulates antigen-presenting cells such as dendritic cells, resulting in the upregulation of costimulatory molecules and the production of various inflammatory cytokines required for CD4(+) T cell priming in vivo. However, CD40L- or CD40-deficient mice challenged with viruses mount protective CD4(+) T cell responses that produce normal levels of interferon gamma, suggesting a CD40/CD40-independent mechanism of CD4(+) T cell priming that to date has not been elucidated. Here we show that CD4(+) T cell responses to viral infection were greatly diminished in CD40-deficient mice by administration of a soluble form of TNF. ***related*** activation-induced cytokine receptor (TRANCE-R) to inhibit the function of another TNF family member, TRANCE. Thus, the TRANCE/TRANCE-R interaction provides costimulation required for efficient CD4(+) T cell priming during viral infection in the absence of CD40L/CD40. These results also indicate that not even the potent inflammatory microenvironment induced by viral infections is sufficient to elicit efficient CD4(+) T cell priming without proper costimulation provided by the TNF family (CD40L or TRANCE). Moreover, the data suggest that TRANCE/TRANCE-R may be a novel and important target for immune intervention.

L4 ANSWER 5 OF 11 MEDLINE

AN 1999175482 MEDLINE
 DN 99175482
 TI Identification of a new member of the ***tumor***
 necrosis
 factor ***family*** and its receptor, a human ortholog of mouse GITR.
 AU Gurney A L, Masters S A, Huang R M, Pitti R M, Mark D T, Baldwin D T, Gray A M, Dowd A D, Brush A D, Heldens A D, Schow A D, Goddard A D, Wood W I, Baker K P, Godowski P J, Ashkenazi A
 CS Department of Molecular Biology Genetics Inc. 1 DNA Way South San Francisco California 94080 USA
 SO CURRENT BIOLOGY. (1999 Feb 25) 9 (4) 215-8.
 Journal code: B44. ISSN: 0960-9822.
 CY ENGLAND: United Kingdom
 DT Journal, Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199906
 EW 19990604
 AB The tumor necrosis factor (TNF) and TNF receptor (TNFR) gene superfamilies regulate diverse biological functions, including cell proliferation, differentiation, and survival [1] [2] [3]. We have identified a new TNF. ***related*** ligand, designated human GITR ligand (hGITRL), and its human receptor (hGITR), an ortholog of the recently discovered murine glucocorticoid-induced TNFR. ***related*** (mGITR) protein [4]. The hGITRL gene mapped to chromosome 1q23, near the gene for the TNF homolog Fas/CD95 ligand [5]. The hGITR gene mapped to chromosome 1p36, near a cluster of five genes encoding TNFR homologs [1] [6]. We found hGITRL mRNA in several peripheral tissues, and detected hGITRL protein on cultured vascular endothelial cells. The levels of hGITR mRNA in tissues were generally low, in peripheral blood T cells, however, antigen-receptor stimulation led to a substantial induction of hGITR transcripts. Coexpression of hGITRL and hGITR in embryonic kidney 293 cells activated the anti-apoptotic transcription factor NF-kappaB, via a pathway that appeared to involve TNFR-associated factor 2 (TRAF2) [7] and NF-kappaB-inducing kinase (NIK) [8]. Coexpression of hGITRL and hGITR in Jurkat T leukemia cells inhibited antigen-receptor-induced cell death. Thus, hGITRL and hGITR may modulate T lymphocyte survival in peripheral tissues.

peripheral tissues.
 L4 ANSWER 6 OF 11 MEDLINE
 AN 1999128078 MEDLINE
 DN 99128078
 TI Human astrocytic brain tumors express APO2L/TRAIL.
 AU Rieger J, Ohgaki H, Kleihues P, Weller M
 CS Department of Molecular Neurology, University of Tübingen, Germany.
 SO ACTA NEUROPATHOLOGICA. (1999 Jan) 97 (1) 1-4.
 Journal code: ICE. ISSN: 0001-6322.
 CY GERMANY: Germany, Federal Republic of
 DT Journal, Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199908
 EW 19990804
 AB APO2 ligand (APO2L) is a CD95 ligand (CD95L). ***related*** cytokine of the ***tumor*** ***necrosis*** ***factor*** ***family*** that interacts with agonistic (DR4, DR5) and antagonistic (DcR1, DcR2) receptors. Cultured malignant glioma cells preferentially express agonistic receptors and are susceptible to APO2L-induced apoptosis. Here, we report that 8 of 8 human glioma cell lines expressed APO2L mRNA and protein in vitro. Immunohistochemistry using a monoclonal antibody to APO2L revealed that all 23 primary astrocytic brain tumors analyzed, including low-grade astrocytomas and glioblastomas, express APO2L in vivo. With the exception of reactive astrocytes, non-neoplastic glia and neurons in the cerebrum lacked immunoreactivity of APO2L. Thus, in addition to the CD95/CD95L system, a second death ligand/death receptor pair may regulate susceptibility to apoptosis in human glial neoplasms.

L4 ANSWER 7 OF 11 MEDLINE
 AN 1999003284 MEDLINE
 DN 99003284
 TI Interleukin-1 protects transformed keratinocytes from tumor necrosis factor- ***related*** apoptosis-inducing ligand
 AU Kohny-Wilkes G, Kulms D, Poppelmann B, Luger T A, Kubin M, Schwarz T
 CS Department of Dermatology, Ludwig Boltzmann Institute for Cell Biology and Immunobiology of the Skin, University of Münster, Von-Esmarckstrasse 56, D-48149 Münster, Germany.
 SO JOURNAL OF BIOLOGICAL CHEMISTRY. (1998 Oct 30)

273 (44) 29247-53.
 Journal code: HIV. ISSN: 0021-9258.
 CY United States
 DT Journal, Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals; Cancer Journals
 EM 19990204
 EW 19990204
 AB Tumor necrosis factor- ****related**** apoptosis-inducing ligand (TRAIL)
 is a member of the ****tumor**** ****necrosis****
 ****factor****
 ****family****. It induces apoptosis primarily of transformed but not of normal cells and may therefore be a promising anti-cancer drug. Studying the role of TRAIL in apoptosis of keratinocytes, we detected TRAIL transcripts and protein in both normal human keratinocytes and transformed keratinocyte cell lines HaCaT and KB. Although normal keratinocytes were resistant to TRAIL, HaCaT and KB cells underwent apoptosis following TRAIL exposure. When HaCaT and KB cells were pretreated with the pro-inflammatory cytokine interleukin-1 (IL-1), cells became resistant to TRAIL-induced apoptosis. IL-1 significantly induced activation of the transcription factor NF-kappaB in transformed keratinocytes. Moreover, the proteasome inhibitor MG132, which inhibits IL-1-induced NF-kappaB activation, completely prevented the protective effect of IL-1. Thus, IL-1 appears to protect transformed keratinocytes from the cytotoxic effect of TRAIL via activation of NF-kappaB. These data suggest that NF-kappaB activation may protect cells from TRAIL-induced apoptosis and indicate a TRAIL receptor-independent pathway, which allows cells to escape the cytotoxic effect of TRAIL. Because IL-1 is secreted by a variety of tumor cells and is also released by inflammatory cells participating in the tumor-host immune response, tumors under these conditions could become resistant to TRAIL.

L4 ANSWER 8 OF 11 MEDLINE
 AN 1998288312 MEDLINE
 DN 98288312
 TI ERICE, a novel FLICE-activatable caspase.
 AU Humke E W; Ni J; Dixit V M
 CS Department of Cellular and Molecular Biology, University of Michigan

Medical School, Ann Arbor, Michigan 48109, USA.
 NC R01 AG13671 (NIA)
 ST32 GM07863-16 (NIGMS)
 SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Jun 19)
 273 (25) 15702-7
 Journal code: HIV. ISSN: 0021-9258.
 CY United States
 DT Journal, Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals; Cancer Journals
 OS GENBANK-AF078533
 EM 199809
 AB Programmed cell death, or apoptosis, is a process of fundamental importance to cellular homeostasis in metazoan organisms (Ellis, R. E., Yun, J., and Horvitz, H. R. (1991) Annu. Rev. Cell Biol. 7, 663-698). The caspase family of mammalian proteases, ****related**** to the nematode death protein CED-3, plays a crucial role in apoptosis and inflammation. We report here the isolation and characterization of a new caspase, tentatively termed ERICE (Evolutionarily ****Related**** Interleukin-1beta Converting Enzyme). Based on phylogenetic analysis, ERICE (caspase-13) is a member of the ICE subfamily of caspases which includes caspase-1 (ICE), caspase-4 (ICE1), TX, ICH-2, and caspase-5 (ICE1-IL, TY). Overexpression of ERICE induces apoptosis of 293 human embryonic kidney cells and MCF7 breast carcinoma cells. Like other members of the subfamily, ERICE is not activated by the serine protease granzyme B, a caspase-activating component of cytotoxic T cell granules. Therefore, ERICE most likely does not play a role in granzyme B-induced cell death. ERICE, however, was activated by caspase-8 (FLICE, MACH, Mch-5), the apical caspase activated upon engagement of death receptors belonging to the ****tumor**** ****necrosis**** ****factor****
 ****family****. This is consistent with a potential role for ERICE in this receptor-initiated death pathway.

L4 ANSWER 9 OF 11 MEDLINE
 AN 1998269066 MEDLINE
 DN 98269066
 TI Molecular mechanisms of promoter regulation of the gp34 gene that is trans-activated by an oncoprotein Tax of human T cell leukemia virus type I.
 AU Ohnani K; Tsujimoto A; Tsubakara T; Numata N; Miura S;

Suganuma K;
 Nakamura M
 CS Human Gene Sciences Center, Japan
 SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Jun 5) 273 (23) 14119-29.
 Journal code: HIV. ISSN: 0021-9258.
 CY United States
 DT Journal, Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals; Cancer Journals
 OS GENBANK-AB007839
 EM 19980903
 EW 19980903
 AB We investigated the molecular mechanism of transcriptional activation of the gp34 gene by the Tax oncoprotein of human T cell leukemia virus type I (HTLV-I). gp34 is a type II transmembrane molecule belonging to the ****tumor**** ****necrosis**** ****factor****
 ****family**** and is constitutively expressed on HTLV-I-producing cells but not normal resting T cells. The transcriptional regulatory region of the gp34 gene was activated by HTLV-I Tax in the human T cell line Jurkat, in which endogenous gp34 is induced by Tax. Sequence analysis demonstrated that two NF-kappaB-like elements (1 and 2) were present in the regulatory region. Both NF-kappaB-like elements were able to bind to NF-kappaB or its ****related**** factor(s) in a Tax-dependent manner. Chromatinol acetyltransferase assays indicated that NF-kappaB-like element 1 was Tax-responsive, although the activity was lower than that the native promoter. NF-kappaB-like element 2 elevated promoter activity when combined with NF-kappaB-like element 1, indicating cooperative function of the elements for maximum promoter function. Unlike typical NF-kappaB elements, the NF-kappaB-like elements in gp34 were not activated by treatment of Jurkat cells with phorbol ester despite induction of the NF-kappaB-like binding activity. Chromatinol acetyltransferase reporter assays using the region upstream of the NF-kappaB-like elements identified an upstream region that reduced transcription from cognate and noncognate core promoters in a Tax-independent manner. Our results imply complex regulation of expression of the gp34 gene and suggest implication of gp34 in proliferation of HTLV-I infected T cells.

L4 ANSWER 10 OF 11 MEDLINE
 AN 199803918 MEDLINE
 DN 9803918
 TI Apoptotic signaling in lymphocytes.
 AU Rudin C M, Van Dongen J, Thompson C B
 CS Gwenn Knapp Center for Lupus and Immunology Research, University of Chicago, IL 60637-5420, USA
 SO CURRENT OPINION IN HEMATOLOGY, (1996 Jan) 3 (1) 35-40. Ref: 28
 Journal code: CNO. ISSN: 1065-6251.
 CY United States
 DT Journal, Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LA English
 FS Priority Journals
 EM 199802
 EW 19980204
 AB Two families of cell surface receptors are integral to the control of lymphocyte survival and programmed cell death (apoptosis): the tumor necrosis factor receptor family and the CD28/CTLA4 family. Tumor necrosis factor receptor family members bind a ***related*** collection of ligands (the ***tumor*** **nerosis*** **factor*** **family***) that can either induce or inhibit cell death. Two of the tumor necrosis factor receptor family members, tumor necrosis receptor 1 and Fes, have been implicated in the termination of immune responses through their ability to induce apoptosis. A number of cytoplasmic proteins implicated in signal generation by these receptors recently have been identified. These proteins fall into several ***related*** classes sharing intriguing structural motifs. The CD28 and CTLA4 molecules share at least two extracellular ligands and signaling through the two receptors appears to determine the apoptotic sensitivity of activated T cells. The effects of CD28 and CTLA4 on cell survival are dependent on T-cell antigen receptor engagement, providing a potent mechanism for clonally specific T-cell expansion or deletion. The study of the apoptotic pathways in lymphocytes has led to a better understanding of the mechanisms of autoimmune disease and serves as a model system for the study of the regulation of cell survival and tissue homeostasis.

AN 97390509 MEDLINE
 DN 97390509
 TI Control of TRAIL-induced apoptosis by a family of signaling and decoy receptors [see comments]
 CM Comment in: Science 1997 Aug 8;277(5327):768
 AU Sheridan J P, Masters S A, Pitti R M, Gurney A, Skubatch M, Baldwin D, Ramakrishnan L, Gray C L, Baker K, Wood W J, Goddard A D, Godowski P, Ashkenazi A
 CS Department of Molecular Oncology, Genentech, South San Francisco, CA 94080-4918, USA
 SO SCIENCE, (1997 Aug 8) 277 (5327) 818-21.
 Journal code: UJ7. ISSN: 0036-8075.
 CY United States
 DT Journal, Article; (JOURNAL ARTICLE)
 LA English
 FS Cancer Journals; Priority Journals
 OS GENBANK-AF012535; GENBANK-AF012536
 EM 199710
 AB TRAIL (also called Apo2L) belongs to the ***tumor*** **nerosis*** **factor*** **family*** , activates rapid apoptosis in tumor cells, and binds to the death-signaling receptor DR4. Two additional TRAIL receptors were identified. The receptor designated death receptor 5 (DR5) contained a cytoplasmic death domain and induced apoptosis much like DR4. The receptor designated decoy receptor 1 (DcR1) displayed properties of a glycopospholipid-anchored cell surface protein. DcR1 acted as a decoy receptor that inhibited TRAIL signaling. Thus, a cell surface mechanism exists for the regulation of cellular responsiveness to pro-apoptotic stimuli.

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 FILE MEDLINE ENTERED AT 10:33:58 ON 10 JUL 2000
 L1 1 S TRELL/AB,BI
 L2 0 S TUMOR NECROSIS FACTOR FAMILY RELATED PROTEIN#/AB,BI
 L3 53 S TUMOR NECROSIS FACTOR FAMILY Y/AB,BI
 L4 11 S L3 AND RELATED/AB,BI
 FILE MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS ENTERED AT 10:39:33 ON 10 JUL 2000
 ==> s 11 or 12
 'AB' IS NOT A VALID FIELD CODE
 4 FILES SEARCHED ...
 L5 9 L1 OR L2
 ==> dup rem 15
 PROCESSING COMPLETED FOR L5
 L6 8 DUP REM L5 (1 DUPLICATE REMOVED)
 ==> d 1 - bib ab
 YOU HAVE REQUESTED DATA FROM 8 ANSWERS - CONTINUE? Y(N)y
 L6 ANSWER 1 OF 8 INPADOC COPYRIGHT 2000 EPO LEVEL 1
 AN 127892689 INPADOC ED 20000523 EW 200020 UP 20000523 UW 200020
 TI LIGANDO RELACIONADO A FACTOR DE NECROSE DE TUMOR
 IN YVES CHICHEPORTICHE; JEFFREY L. BROWNING INS CHICHEPORTICHE YVES; BROWNING JEFFREY L PA BIOGEN, INC.; BIOGEN, INC.; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA
 GENEVA, THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA
 PAS BIOGEN INC; FACULTY OF MEDICINE OF THE UNI PAA US; CH
 DT Patent

PTI BRA UNEXAMINED PATENT APPLICATION
PI BR 9711046 A 20000111
AI BR 1997-11046 A 19970807
PRAI US 1996-23541 P 19960807
US 1996-28515 P 19961018
US 1997-40820 P 19970318
WO 1997-US13945 W 19970807
AB Patente de Invenção: LIGANDO RELACIONADO A
FATOR E NECROSE DE
TUMOR<D> Lígando relacionado a fator de necrose de tumor (TNF), ***TRELL*** modificado, e composto as farmas utises compreendendo os mesmos.

L6 ANSWER 2 OF 8 INPADOC COPYRIGHT 2000 EPO
LEVEL 2
AN 44303990 INPADOC EW 199923 UW 199926
TI TUMORNEKROSEFAKTOR-RELATERT LIGAND (***TRELL***) ET NYTTI MEDLEM AV TUMORNEKROSEFAKTORFAMILIEN (TNF), MODIFISERT ***TRELL*** OG FARMAS YTISKE SAMMENSETNINGER INNEHOLDENDE SLIKE IN CHICHEPORTICHE, YVES, BROWNING, JEFFREY L. INS CHICHEPORTICHE YVES, BROWNING JEFFREY L. INA CH US
PA BIOGEN INC
PAS BIOGEN INC
PAA US
DT Patent
PIT NOAO APPLICATION FILED
PI NO 9900350 A0 19990205
AI NO 1999-550 A 19990205
PRAI US 1996-23541 P 19960807
US 1996-28515 P 19961018
US 1997-40820 P 19970318
WO 1997-US13945 W 19970807

L6 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1998-112459 CAPLUS
DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a tumor necrosis factor-related ligand or its receptor
IN Chicheportiche, Yves; Browning, Jeffrey L.
PA Biogen, Inc., USA; Faculty of Medicine of the University of Geneva.
Chicheportiche, Yves; Browning, Jeffrey L.
SO PCT Int. Appl., 69 pp.
CODEN: PDXXD2
DT Patent
LA English
FANCNT 1
PATENT NO. KIND DATE APPLICATION NO.
DATE

PI WO 9805783 A1 19980212 WO 1997-US13945
19970807
W. AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW, GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BE, BI, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
AU 9738294 A1 19980225 AU 1997-38294 19970807
CN 1232503 A 19991020 CN 1997-198401 19970807
EP 956351 A1 19991117 EP 1997-935334 19970807
R. AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LT, LU, NL, SE, MC, PT, IE, SL, LT, LV, FI, RO
BR 9711046 A 20000111 BR 1997-11046 19970807
NO 9900350 A 19990406 NO 1999-550 19990205
PRAI US 1996-PV23541 19960807
US 1996-PV28515 19961018
US 1997-PV40820 19970318
WO 1997-US13945 19970807
AB Tumor necrosis factor-related ligand (***TRELL***), a novel member of the tumor necrosis factor family (TNF), modified ***TRELL*** , and pharmaceutical compns. comprising them. The ***TRELL*** protein or its receptor may have anti-cancer and/or immunoregulatory applications. Human cells transfected with the ***TRELL*** gene may be used in gene therapy to treat tumors, autoimmune and inflammatory disease or inherited genetic disorders. ***TRELL*** -specific monoclonal antibodies and antisense RNA against ***TRELL*** are also claimed. The method, is exemplified by treating human adenocarcinoma cells with ***TRELL*** or ***TRELL*** homologs.

L6 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997-69723 BIOSIS
DN PREVI9799796436
TI Morphobiochemical studies of Furcraea (Agraceae) of India.
AU Khan, Hafiz Ahmed
CS Birlal Sahni Inst. Palaeobotany, 53 University Rd., Lucknow 226007 India
SO Journal of Plant Anatomy and Morphology (Iodipur), (1997) Vol. 7, No. 2.

pp. 140-147.
ISSN: 0256-436X
DT Article, (TAXONOMIC KEY)
LA English
AB Few species of Furcraea Vent. have been introduced in India as garden and hedge plants, and for obtaining fibre. These are succulent plants like Agave and are growing in dry, tropical and subtropical places throughout the country. F. gigantea Vent. is a common species and a more important plant known as Mauritius Hemp. Other species grown in India are F. bedinghausii Koch, F. longerva Karw. & Zucc. F. sellos C. Koch. var. marginata ***Trell***, and F. hexapetala Urb. The botanical identity of south Indian species known as Mauritius Hemp is F. hexapetala Urb. (Syn. F. cubensis Haw.) and not F. gigantea Vent. The F. gigantea is a large shrub with fleshy leaves possessing a brown tip spine and armed or often Basal part only, armed margins. Trunk is long below the rosette of leaves. A variety of F. gigantea is mediopetala which is variegated with butter coloured straps along the leaves. This variety is generally grown as ornamental in the gardens in pots or on the ground. Leaves of willmetiana, the other variety are light green coloured, armed with prickles and the juice is of mild odour. Variety marginata of F. sellos has the leaf margins armed with distant brown horny hooked prickles.

L6 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2000 ACS
AN 1991-141403 CAPLUS
DN 114:141403
TI Meningococcal class I outer-membrane protein vaccine
IN Seid, Robert C., Jr.; Paradiso, Peter R.; Poolman, Jan T.; Hoogthout, Peter; Wientz, Emmanuel J. H. J.; Van der Ley, Peter; Heckels, John
Edward, Clarke, Jan Nicholas
PA Praxis Biologics, Inc., USA; Rijksinstituut voor Volksgezondheid en Miliehygiene
SO PCT Int. Appl., 121 pp.
CODEN: PDXXD2
DT Patent
LA English
FANCNT 1
PATENT NO. KIND DATE APPLICATION NO.
DATE

P1 WO 9006696 A2 19900628 WO 1989-US5678 19891219
 WO 9006696 A3 19900712
 W: AU, DK, FI, JP, NO, US
 RW: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE
 NL 8803111 A 19900716 NL 1988-3111 19881219
 NL 8900036 A 19900716 NL 1989-36 19890106
 NL 8901612 A 19900716 NL 1989-1612 19890626
 AU 9048219 A1 19900710 AU 1990-48219 19891219
 AU 640118 B2 19930819
 EP 449958 A1 19911009 EP 1990-901397 19891219
 EP 449958 B1 19950322
 R: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE
 JP 06503465 T2 19940421 JP 1990-501662 19881219
 AT 120093 E 19950415 AT 1990-901397 19891219
 ES 2070312 T3 19950601 ES 1990-901397 19891219
 CA 2007248 AA 19900706 CA 1990-2007248 19900105
 NO 9102369 A 19910806 NO 1991-2369 19910618
 DK 9101174 A 19910815 DK 1991-1174 19910618
 PRAI NL 1988-3111 19881219
 NL 1989-30 19890106
 NL 1989-1612 19890626
 NL 1989-36 19890106
 WO 1989-US5678 19891219
 AB Outer-membrane vesicles, class 1 outer-membrane proteins (OMPs) of
 Neisseria meningitidis, fragments or oligopeptide congl. epitopes of
 the class 1 OMPs, and antigenic conjugates are provided for
 immunization
 against meningococcal disease. Also provided are cloning and
 prodn. of
 fusion proteins congl. class 1 OMP epitopes and flagellin protein.
 Epitope sequences are identified, and DNA sequencing of class 1
 OMP genes
 from different N. Meningitidis serotypes is presented. Thus,
 recombinant flagellins congl. either a VR1 (1st variable region of
 class 1
 OMP), VR2, or a cassette of both VR1 and VR2 are effective in
 eliciting
 antibody response which was cross-reactive to purified P1.16 (class
 1 OMP
 subtype) and, to a lesser extent, to outer-membrane complex. Each
 construction also induced significant anti-flagellin titers; control
 wild
 type flagellin only induced antibody response to flagellin itself.
 Recombinant flagellin-oligosaccharide conjugate also proved and
 tested

S0 Scandinavian Journal of Primary Health Care, (1984) 2/3 (96-97).
 CODEN: SJPCD7
 CY Sweden
 DT Journal
 FS 022 Human Genetics
 017 Public Health, Social Medicine and Epidemiology
 018 Cardiovascular Diseases and Cardiovascular Surgery
 006 Internal Medicine
 LA English
 AB ***Trell*** and collaborators have tried to define the
 hypertensive
 genotype by an analysis of risk factors in hypertensive patients with
 genotype by an analysis of risk factors in hypertensive patients with
 a
 varying degree of genetic predisposition. The data support the view
 that
 the genetic predisposition for hypertension is not per se associated
 with
 such accepted cardiovascular risk factors in the population as high
 serum
 cholesterol and triglyceride content, and impaired glucose
 tolerance. What
 is this polygenic predisposition to hypertension like? Gradually it
 has
 proved possible to define some contributing factors. Increased
 sensitivity
 to sodium loading - the mechanism known to be active in some
 strains of
 rats - may result in hypertension in humans as well. An elevated
 intracellular sodium concentration with increased smooth muscle
 reactivity has been demonstrated in hypertensive patients. Data
 are in
 existence supporting a correlation between hypertension and a
 number of
 varying traits: Certain HLA-alleles, the C3F-allele in the
 complement
 system, different autoantibodies, herpesvirus antibodies, increased
 adrenal responsiveness to angiotensin-II, increased catecholamine
 release
 during exercise, a high proportion of fast twitch fibres in skeletal
 muscles. Probably this spectrum of characteristics will be further
 broadened in the future. The genetic predisposition to hypertension
 must
 be considered the result of the presence or absence of these traits.
 The
 person who, at the same time, is salt sensitive, C3F positive, with a
 high
 proportion of fast twitch muscle fibres, etc is particularly
 predisposed.
 Today it is not possible to single out the relative importance of
 individual factors in the pathogenesis of human hypertension. Nor
 can we
 predict to what extent a diagnostic disentanglement along these
 lines
 should determine the therapeutic strategy.

DN BA73:34902
 TI HIRSUTINOLIDES FROM VERNONIA-SP.
 AU BOHLMANN F, MUELLER L, GUPTA R K, KING R M, ROBINSON H
 CS INST ORG CHEM, TECHNICAL UNIV, BERLIN, D-1000
 BERLIN 12, W, GER
 SO PHYTOCHEMISTRY (OXF), (1981) 20 (9), 223-2238.
 CODEN: PTICAS, ISSN: 0031-9422
 FS BA, OLD
 LA English
 AB Of the 19 spp. of Vernonia [V. alameda H. Robins., V. condensata Baker, V. coriacea Less., V. echinifolia Mart., V. farinosa Baker, V. gigantea ***Trell***, Branner et Cor., V. hegel H. Robins., V. holosericea Mart. ex DC, V. intermedia DC, V. kunzei Hieron., V. laxa Gardn., V. maritima Mart., V. missionis Gardn., V. myrsinitis Ekman, V. obtusata Less., V. regis H. Robins., V. reixeciae H. Robins., V. tomentella Mart. and V. venosissima Sch. Bip. ex Baker] studied, 5 contained highly oxygenated sesquiterpene lactones; the rest contained predominantly triterpenes, especially lupane derivatives.

L6 ANSWER 8 OF 8 MEDLINE DUPLICATE 1
 AN 76058643 MEDLINE
 DN 76058643
 TI Hydanion derivatives and malignancies of the haemopoietic system.
 AU Bichel J
 SO ACTA MEDICA SCANDINAVICA, (1975 Oct) 198 (4) 327-8.
 Journal code: 14G, ISSN: 0001-6101.
 CY Sweden
 DT Journal, Article, (JOURNAL ARTICLE)
 LA English
 FS Abstracted Index Medicus Journals, Priority Journals
 EM 197603
 AB Two patients are described who developed malignant lymphoma (lymphosarcoma) after diphenylhydantoin therapy because of epilepsy.
 Malignant lymphoma in a few patients receiving this medication has been described earlier. The literature has been reviewed and discussed recently
 by Rausing and ***Trell*** (2).

=> e browning /au
 E1 1 BROWNING ILEY B/AU
 E2 6 BROWNING IVAN/AU
 E3 203 -> BROWNING I/AU
 E4 59 BROWNING J A/AU
 E5 2 BROWNING J ARTE/AU

E6 2 BROWNING J B/AU
 E7 5 BROWNING J C/AU
 E8 103 BROWNING J D/AU
 E9 38 BROWNING J E/AU
 E10 3 BROWNING J F/AU
 E11 28 BROWNING J G/AU
 E12 3 BROWNING J H/AU

 => s c3-c5
 L7 264 (BROWNING J/AU OR BROWNING J A/AU OR BROWNING J AKTIE/AU)
 => e browning,jeffrey/au
 E1 1 BROWNING JEFFREY/AU
 E2 1 BROWNING JEFFREY L/AU
 E3 37-> BROWNING JEFFREY/AU
 E4 2 BROWNING JEFFREY C/AU
 E5 1 BROWNING JEFFREY CHARLES/AU
 E6 1 BROWNING JEFFREY D/AU
 E7 1 BROWNING JEFFREY J/AU
 E8 146 BROWNING JEFFREY L/AU
 E9 2 BROWNING JEFFREY LEE/AU
 E10 1 BROWNING JENNIFER L/AU
 E11 1 BROWNING JENNIFER L/AU
 E12 1 BROWNING JENNIFER S/AU

 => s c1-c9
 L8 192 (BROWNING JEFFREY/AU OR BROWNING JEFFREY L/AU OR BROWNING JEFFREY/AU OR BROWNING JEFFREY C/AU OR BROWNING JEFFREY CHARLES/AU OR BROWNING JEFFREY D/AU OR BROWNING JEFFREY J/AU OR BROWNING JEFFREY L/AU OR BROWNING JEFFREY LEE/AU)
 => s i7 or i8
 L9 456 L7 OR L8
 => s i9 and tumor necrosis factor/ab,bi
 /AB IS NOT A VALID FIELD CODE
 L10 74 L9 AND TUMOR NECROSIS FACTOR/AB,BI
 => s i10 and family/ab,bi
 /AB IS NOT A VALID FIELD CODE
 L11 26 L10 AND FAMILY Y/AB,BI
 => dup rem i11
 PROCESSING COMPLETED FOR L11
 L12 16 DUP REM L11 (10 DUPLICATES REMOVED)

=> d i - bib ab
 YOU HAVE REQUESTED DATA FROM 16 ANSWERS - CONTINUE? Y/(N)/y
 L12 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2000 ACS AN 1999-329685 CAPLUS DN 131:115182
 TI Signaling through the lymphotoxin- β receptor stimulates HIV-1 replication alone and in cooperation with soluble or membrane-bound
 TNF- α
 AU Marshall, William L.; Brinkman, Brigitta M. N.; Ambrose, Christine M.; Pesavento, Patricia A.; Ugialoro, Adele M.; Teng, Edna; Finberg, Robert
 W.; ***Browning, Jeffrey L.***; Goldfield, Anne E.
 CS Division of Adult Oncology, Dana-Farber Cancer Institute, Boston, MA.
 02115, USA
 SO J. Immunol. (1999), 162(10), 6016-6023
 CODEN: JOLMAJ; ISSN: 0022-1767
 PB American Association of Immunologists
 DT Journal
 LA English
 AB The level of ongoing HIV-1 replication within an individual is crit. to HIV-1 pathogenesis. Among host immune factors, the cytokine TNF- α has previously been shown to increase HIV-1 replication in various monocyte and T cell model systems. Here, the authors demonstrate signaling through the TNF receptor ***family*** member, the lymphotoxin- β (LT- β) receptor (LT- β R), also regulates HIV-1 replication. Furthermore, HIV-1 replication is cooperatively stimulated when the distinct LT- β R and TNF receptor systems are simultaneously engaged by their specific ligands. Moreover, in a physical coculture cellular assay system, the authors show that membrane-bound TNF- α and LT- α 1 beta 2 act virtually identically to their sol. forms in the regulation of HIV-1 replication. Thus, co-signaling via the LT- β R and TNF- α receptors is probably involved in the modulation of HIV-1 replication and the subsequent detn. of HIV-1 viral burden in monocytes. Intriguingly, surface expression of LT- α 1 beta 2 is up-regulated on a T cell line acutely infected with HIV-1, suggesting a pos. feedback loop between HIV-1 infection, LT- α 1 beta 2 expression, and HIV-1

replication. Given the crit. role that LT- α 1 beta 2 plays in lymphoid architecture, the authors speculate that LT- α 1 beta 2 may be involved in HIV-assoc. abnormalities of the lymphoid organs.
 RE CNT 65
 RE
 (1) Amodio, A. Immunol Today 1990, P374 CAPLUS
 (2) Balder, M. Science 1996, V274, P1464 CAPLUS
 (3) Bazzoni, F. J Inflamm 1995, V45, P221 CAPLUS
 (4) Bergelson, J. Science 1992, V255, P1718 CAPLUS
 (5) Bonissio, V. Proc Natl Acad Sci USA 1994, V91, P7007 CAPLUS
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L12 ANSWER 2 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 1 AN 1999-325998 BIOSIS DN PREV199900325998
 TI BAF, a novel ligand of the ***tumor*** ***necrosis*** ***factor*** ***family***, stimulates B cell growth.
 AU Schneider, Pascal; Mackay, Fabienne; Steiner, Veronique; Hofmann, Kay; Bodmer, Jean-Luc; Holler, Nils; Ambrose, Christine; Lawton, Pernis; Bixler, Sarah; Achia-Obea, Hans; Valmori, Daniel; Romero, Pedro; Werner-Favre, Christine; Zuber, Rudolph H.; ***Browning, Jeffrey L.***
 ; Tschopp, Jung (1)
 CS (1) Institute of Biochemistry, University of Lausanne, CH des Boveresses 155, CH-1066, Epalinges Switzerland
 SO Journal of Experimental Medicine, (June 11, 1999) Vol. 189, No. 11, pp. 1747-1756.
 ISSN: 0022-1007.
 DT Article
 LA English
 SL English
 AB Members of the ***tumor*** ***necrosis*** ***factor*** (TNF) ***family*** induce pleiotropic biological responses, including cell growth, differentiation, and even death. Here we describe a novel member of the TNF ***family***, designated BAF (for B cell activating factor belonging to the TNF ***family***), which is expressed by T cells and dendritic cells. Human BAF was mapped to chromosome 13q32-34. Membrane-bound BAF was processed and secreted through the action of a protease whose specificity matches that of the furin ***family*** of proprotein convertases. The expression of BAF receptor appeared to be restricted to B cells. Both membrane-bound and soluble BAF

induced proliferation of anti-immunoglobulin M-stimulated peripheral blood B lymphocytes. Moreover, increased amounts of immunoglobulins were found in supernatants of germinal center-like B cells costimulated with BAF. These results suggest that BAF plays an important role as costimulator of cell proliferation and function.

L12 ANSWER 3 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 2
AN 2000.50715 BIOSIS
DN PREV20000030715
TI Mice transgenic for BAF develop lymphocytic disorders along with autoimmune manifestations.

AU Mackey, Fabienne (1); Woodcock, Stephen A.; Lawton, Pomati; Anthrose, Christine; Baetscher, Manfred; Schneider, Pascal; Tschopp, Jurg; ***Browning, Jeffrey L.***
CS (1) Biogen, 12 Cambridge Center, Cambridge, MA USA
SO Journal of Experimental Medicine, (Dec. 6, 1999) Vol. 190, No. 11, pp. 1697-1710.
ISSN: 0022-1007.

DT Article
LA English
SL English
AB The cause of many autoimmune and inflammatory diseases is unresolved, although dysregulated production of ***tumor*** necrosis factor*** (TNF) ***family*** members appears to be important in many cases. BAF, a new member of the TNF ***family***, binds to B cells and costimulates their growth in vitro. Mice transgenic for BAF have vastly increased numbers of mature B and effector T cells, and develop autoimmune-like manifestations such as the presence of high levels of rheumatoid factors, circulating immune complexes, anti-DNA autoantibodies, and immunoglobulin deposition in the kidneys. This phenotype is reminiscent of certain human autoimmune disorders and suggests that dysregulation of BAF expression may be a critical element in the chain of events leading to autoimmunity.

L12 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2000 ACS
AN 1998.112459 CAPLUS
DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a ***tumor*** necrosis factor*** related ligand

or its receptor

IN Chicheportiche, Yves; ***Browning, Jeffrey L.***
PA Biogen, Inc., USA; Faculty of Medicine of the University of Geneva,
Chicheportiche, Yves; Browning, Jeffrey L.
SO PCT Int. Appl. 69 pp.
CODEN: PLOXDD

DT Patent
LA English
FAN/CNT 1
PATENT NO. KIND DATE APPLICATION NO.
DATE

PI WO 9805783 A1 19980212 WO 1997-US13945
19970807
W. AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ,
LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US,
UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR,
GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA,
GN, ML, MR, NE, SN, TD, TG

AU 9738294 A1 19980225 AU 1997-38294 19970807
CN 1232503 A 19991020 CN 1997-198401 19970807
EP 956351 A1 19991117 EP 1997-95334 19970807
R. AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO
BR 9711046 A 20000111 BR 1997-11046 19970807
NO 9900550 A 19990406 NO 1999-550 19990205
PRAI US 1996-PV23541 19960807
US 1996-PV23515 19961018
US 1997-PV40820 19970318
WO 1997-US13945 19970807

AB ***Tumor*** necrosis factor*** related ligand (TRELL), a novel member of the ***tumor*** necrosis factor*** family*** (TNF), modified TRELL, and ***factor*** (TNF), modified TRELL, and anti-cancer and/or immunoregulatory applications. Human cells have been transfected with the TRELL gene may be used in gene therapy to treat tumors, autoimmune and inflammatory disease or inherited genetic disorders. TRELL-specific monoclonal antibodies and antisense RNA against TRELL are

also claimed. The method, is exemplified by treating human adenocarcinoma cells with TRELL or TRELL homologs.

L12 ANSWER 5 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 3
AN 1999.37693 BIOSIS
DN PREV19990037693
TI Both the lymphotoxin and ***tumor*** necrosis factor*** pathways are involved in experimental murine models of colitis.

AU Mackey, Fabienne (1); ***Browning, Jeffrey L.***; Lawton, Pomati;
Shah, Samir A.; Comiskey, Martine; Bhan, Anil K.; Mizoguchi, Emiko;
Tenhore, Cox; Simpson, Stephen J.
CS (1) Biogen, 12 Cambridge Center, Cambridge, MA 02142 USA
SO Gastroenterology, (Dec., 1998) Vol. 115, No. 6, pp. 1464-1475.
ISSN: 0016-5085.

DT Article
LA English
AB Background & Aims: Membrane lymphotoxin (LT) alpha/beta, a member of the ***tumor*** necrosis factor*** (TNF) ***family*** of immune regulatory molecules, is involved both in the development of secondary lymphoid tissues and the maintenance of organized lymphoid tissues in the adult. Defects observed in the mucosal immune system in animals with a genetically disrupted LT alpha/beta pathway coupled with the expression of LT alpha/beta in activated T cells motivated an examination of the importance of this pathway in experimental colitis. Methods: Soluble LT beta receptor (LT betaR) immunoglobulin fusion protein was used to inhibit the LT alpha/beta light axis in two independent rodent models of colitis: CD45RBhi CD4+-reconstituted SCID mice and bone marrow transplanted (epsilonpsilon26 mice (BM f/wdarw (epsilonpsilon26). Results: Treatment with LT betaR immunoglobulin attenuated the development of both the clinical and histological manifestations of the disease in these two murine models of colitis. Given the success of TNF inhibitors in the treatment of human Crohn's disease, the effects of LT betaR immunoglobulin have been compared with antibody to TNF in the BM f/wdarw (epsilonpsilon26 model, and both treatments were equally efficacious. Conclusions: The LT pathway plays a role in the development of colitis as important as that of the TNF system and, therefore, represents a potential novel

alter the receptor binding properties of heteromeric complexes assembled from the. Polypeptides comprising LT subunit assocn. domains, LT

heteromeric complexes which inhibit receptor signaling pharmaceutical

comprns comprising LT heteromeric inhibitors, and methods for treatment using those pharmaceutical compns. are also provided

L12 ANSWER 10 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 6

AN 1996:521458 BIOSIS DN PREV199699243814

TI Lymphotoxin beta receptor triggering induces activation of the nuclear

factor kappa-B transcription factor in some cell types.

AU Mackay, Fabienne (1); Majeau, Gerard R.; Hochman, Paula S.; ***Browning ***

*** Jeffrey L. *** CS (1) Dep Cell Biol, Biogen Inc., 12 Cambridge Cent., Cambridge, MA 02142

USA SO Journal of Biological Chemistry, (1996) Vol. 271, No. 40, pp. 24934-24938.

ISSN: 0021-9258. DT Article

LA English

AB NF-kappa-B is a pleiotropic transcription factor capable of activating the expression of a great variety of genes critical for the immunoinflammatory

response. ***Tumor*** ***necrosis*** ***factor*** alpha

(TNF-alpha) and lymphotoxin alpha (LT-alpha, originally TNF-beta) are

potent nuclear factor kappa-B (NF-kappa-B) activators in various cell

types. The LT-alpha molecule, in addition to being secreted as a soluble

trimer, can also form membrane-anchored heterotrimers with the

LT-beta chain, another member of the TNF ***family***. The

LT-alpha-1-beta-2 heterotrimer binds a specific receptor, called the LT-beta receptor

(LT-beta-R), which is also a member of the TNF receptor ***family***

Here, we show that engagement of LT-beta-R with a soluble form of

LT-alpha-1-beta-2 or with a specific anti-LT-beta-R agonistic monoclonal

antibody CBE11 quickly induces activation of NF-kappa-B in HT-29 and WiDr

human adenocarcinomas. LT-beta-R triggering activates NF-kappa-B and

induces proliferation in WI-38 human lung fibroblasts. No NF-kappa-B

activation is observed in human umbilical vein endothelial cells, correlating with the inability of LT-beta-R activation to induce expression of NF-kappa-B-dependent cell surface adhesion molecules. Thus,

like several other members of the TNF receptor ***family***, the

LT-beta-R can activate NF-kappa-B following receptor ligation in some but not all LT-beta-R-positive cells

L12 ANSWER 11 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 7

AN 1996:241639 BIOSIS DN PREV199698789768

TI Preparation and characterization of soluble recombinant heterotrimeric

complexes of human lymphotoxin alpha and beta.

AU ***Browning, Jeffrey L. (1)*** ; Malkowski, Konrad, Griffiths, David

A.; Bourdon, Paul R.; Hession, Catherine; Ambrose, Christine M.; Meier, Werner

CS (1) Biogen, 14 Cambridge Cent., Cambridge, MA 02142 USA SO Journal of Biological Chemistry, (1996) Vol. 271, No. 15, pp. 8618-8626.

ISSN: 0021-9258. DT Article

LA English

AB The lymphotoxin (LT) protein complex is a heteromer of alpha (LT-alpha,

also called ***tumor*** ***necrosis*** ***factor*** (TNF-beta)

and beta (LT-beta) chains anchored to the membrane surface by the transmembrane domain of the LT-beta portion. Both proteins

belong to the TNF ***family*** of ligands and receptors that regulate aspects of the

immune and inflammatory systems. The LT complex is found on activated

lymphocytes and binds to the lymphotoxin-beta receptor, which is generally

present on nonlymphoid cells. The signaling function of this receptor-ligand pair is not precisely known but is believed to be

involved in the development of the peripheral lymphoid organs. To analyze the

properties of this complex, a soluble, biologically active form of the surface complex was desired. The LT-beta molecule was

engineered into a secreted form and co-expressed with LT-alpha using baculovirus/insect cell

technology. By exploiting receptor affinity columns, the

LT-alpha-3, LT-alpha-2/beta-1, and LT-alpha-1/beta-2 forms were purified. All

three molecules were trimers, and their biochemical properties are described.

The level of LT-alpha-3-like components in the LT-alpha-1/beta-2 preparation was found to be 0.02% by following the activity of the preparation in a WEHI 164 cytotoxicity assay. LT-alpha-3 with an asparagine 50 mutation (D50N) cannot bind the TNF receptors.

Heteromeric LT complexes were prepared with this mutant LT-alpha form, allowing a

precise delineation of the extent of biological activity mediated by the

TNF receptors. A LT-alpha-3 based cytotoxic activity was used to show that the LT-alpha-1/beta-2 form cannot readily scramble into a mixture of forms

following various treatments and storage periods. This biochemical characterization of the LT heteromeric ligands and the

demonstration of

their stability provides a solid foundation for both biological studies and an analysis of the specificity of the LT-beta and TNF receptors for

the various LT forms.

L12 ANSWER 12 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 8

AN 1996:229916 BIOSIS DN PREV199698794045

TI Signaling through the lymphotoxin beta receptor induces the death of some

adenocarcinoma tumor lines.

AU ***Browning, Jeffrey L. (1)*** ; Malkowski, Konrad, Sizing, Irene;

Griffiths, David; Zaferi, Mohammad; Benjamin, Christopher D.; Meier,

Werner; Mackay, Fabienne

CS (1) Dep. Immunology/Inflammation, Biogen, 14 Cambridge Center, Cambridge, MA 02142 USA

SO Journal of Experimental Medicine, (1996) Vol. 183, No. 3, pp. 867-878.

ISSN: 0022-1007. DT Article

LA English

AB Surface lymphotoxin (LT) is a heteromeric complex of LT-alpha and LT-beta

chains that binds to the LT-beta receptor (LT-beta-R), a member of the ***tumor*** ***necrosis*** ***factor*** (TNF)

family of receptors. The biological function of this receptor-ligand system is

poorly characterized. Since signaling through other members of this receptor ***family*** can induce cell death, e.g., the TNF and

Fas receptors, it is important to determine if similar signaling events can be

communicated via the LT-beta-R. A soluble form of the surface complex was

produced by coexpression of LT-alpha and a converted form of

L.T-beta
 wherein the normally type II L.T-beta membrane protein was
 changed to a
 type I secreted form. Recombinant L.T-alpha-1/beta-2 was cytotoxic
 to the
 human adenocarcinoma cell lines HT-29, WiDr, MDA-NB-468,
 and HT-3 when
 added with the synergizing agent interferon (IFN) gamma. When
 immobilized
 on a plastic surface, anti-L.T.-std R, monoclonal antibodies (mAbs)
 induced
 the death of these cells, demonstrating direct signaling via the
 L.T-beta-R. Anti-L.T.-beta R mAbs were also identified that
 inhibited
 ligand-induced cell death, whereas others were found to potentiate
 the
 activity of the ligand when added in solution. The human WiDr
 adenocarcinoma line forms solid tumors in immunocompromised
 mice, and
 treatment with an anti-L.T-beta-R antibody combined with human
 IFN-gamma
 arrested tumor growth. The delineation of a biological signaling
 event
 mediated by the L.T.-beta-R opens a window for further studies on
 its
 immunological role, and furthermore, activation of the L.T.-beta-R
 may have
 an application in tumor therapy.

L12 ANSWER 13 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
 DUPLICATE 9
 AN 1994:257127 BIOSIS
 DN PREVI199497270127
 TI A lymphotoxin-beta-specific receptor.
 AU Crowe, Paul D.; Vanarsdale, Todd L.; Walter, Barbara N.; Ware,
 Carl F.
 (1); Hession, Catherine; Elrenfels, Barbara. ***Browning,
 Jeffrey L.***
 ; Din, Wenie S.; Goodwin, Raymond G.; Smith, Craig A.
 CS (1) Div. Biomed. Sci., Univ. Calif., Riverside, CA 92521 USA
 SO Science (Washington D C), (1994) Vol. 264, No. 5159, pp.
 707-710.
 ISSN: 0036-8075.

DT Article
 LA English
 AB ***Tumor*** ***necrosis*** ***factor***. (TNF) and
 lymphotoxin-alpha (L.T-alpha) are members of a ***family***
 of secreted
 and cell surface cytokines that participate in the regulation of
 immune
 and inflammatory responses. The cell surface form of L.T-alpha is
 assembled
 during biosynthesis as a heteromeric complex with
 lymphotoxin-beta
 (L.T-beta), a type II transmembrane protein that is another member
 of the
 TNF ligand ***family***. Secreted L.T.-alpha is a homotrimer

that binds
 to distinct TNF receptors of 60 and 80 kilodaltons; however, these
 receptors do not recognize the major cell surface L.T.-alpha-L.T-beta
 complex. A receptor specific for human L.T.-beta was identified,
 which
 suggests that cell surface L.T. may have functions that are distinct
 from
 those of secreted L.T.-alpha

L12 ANSWER 14 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
 DUPLICATE 10
 AN 1993:273116 BIOSIS
 DN PREVI199396003341
 TI Lymphotoxin beta, a novel member of the TNF ***family***
 that forms a
 heteromeric complex with lymphotoxin on the cell surface.
 AU ***Browning, Jeffrey L. (1)*** ; Ngam-Ek, Apinya (1);
 Lawton, Pomsri
 (1); Demarinis, Janice (1); Tizard, Richard (1); Chow, E.
 Pingchang (1);
 Hession, Catherine (1); O'Brien-Greco, Betsy (1); Foley, Susan F.
 (1);
 Ware, Carl F.
 CS (1) Biogen Incorporated, 14 Cambridge Cent., Cambridge,
 Massachusetts
 02142 USA
 SO Cell. (1993) Vol. 72, No. 6, pp. 847-856.
 ISSN: 0092-8674.

DT Article
 LA English
 AB The lymphokine ***tumor*** ***necrosis***
 factor (TNF)
 has a well-defined role as an inducer of inflammatory responses;
 however,
 the function of the structurally related molecule lymphotoxin
 (L.T-alpha)
 is unknown. L.T.-alpha is present on the surface of activated T, B
 and LAK
 cells as a complex with a 33 kd glycoprotein, and cloning of the
 cDNA
 encoding the associated protein, called lymphotoxin beta (L.T-beta),
 revealed it to be a type II membrane protein with significant
 homology to
 TNF. L.T.-alpha, and the ligand for the CD40 receptor. The gene for
 L.T.-beta
 was found next to the TNF-L.T. locus in the major
 histocompatibility complex
 (MHC), a region of the MHC with possible linkage to autoimmune
 disease.
 These observations raise the possibility that a surface
 L.T.-alpha-L.T.-beta
 complex may have a specific role in immune regulation distinct
 from the
 functions ascribed to TNF.

L12 ANSWER 15 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1993:334815 BIOSIS

DN PREVI199345029540
 TI Lymphotoxin-beta, a new member of the TNF cytokine
 family
 AU Ware, C. (1); Crowe, P.; Van Arsdele, T.; Hession, C.; Tizard,
 R.; Chow,
 P.; ***Browning, J.***
 CS (1) Univ. Calif., Riverside, CA 92521 USA
 SO Journal of Immunology, (1993) Vol. 150, No. 8 PART 2, pp.
 294A.

Meeting Info.: Joint Meeting of the American Association of
 Immunologists
 and the Clinical Immunology Society Denver, Colorado, USA May
 21-25 1993
 ISSN: 0022-1767.

DT Conference
 LA English

L12 ANSWER 16 OF 16 BIOSIS COPYRIGHT 2000 BIOSIS
 AN 1993:286087 BIOSIS
 DN PREVI199345004212
 TI Lymphotoxin-beta: A new member of the TNF ***family***
 that forms a
 heteromeric complex with lymphotoxin on the cell surface.
 AU ***Browning, Jeffrey L. (1)*** ; Tizard, Richard (1);
 Ngam-Ek, Apinya
 (1); Lawton, Pomsri (1); Demarinis, Janice (1); Chow, E.
 Pingchang (1);
 Hession, Catherine (1); Greco, Betsy (1); Foley, Susan (1); Ware,
 Carl F.
 CS (1) Biogen, Cambridge, MA USA
 SO Journal of Cellular Biochemistry Supplement, (1993) Vol. 0, No.
 17 PART B,
 pp. 87.

Meeting Info.: Keystone Symposium on Cytokines and Cytokine
 Receptors:
 From Cloning to the Clinic Keystone, Colorado, USA January
 31-February 7,
 1993
 ISSN: 0733-1959.

DT Conference
 LA English

=> e chichporstich yves/au

E1 1 CHICHOWSKI S/AU
 E2 1 CHICHPORSTICH C/AU
 E3 0-> CHICHPORSTICHE YVES/AU
 E4 1 CHICHTMAN S/AU
 E5 1 CHICHTON A/AU
 E6 3 CHICHU YOSHIE/S/AU
 E7 3 CHICHU A G/AU
 E8 11 CHICHUA A I/AU
 E9 2 CHICHUA B K/AU
 E10 6 CHICHUA D G/AU
 E11 2 CHICHUA D T/AU
 E12 2 CHICHUA DAVID G/AU

=> d his

(FILE HOME ENTERED AT 10:33:47 ON 10 JUL 2000)

FILE MEDLINE ENTERED AT 10:33:58 ON 10 JUL 2000

L1 1 S TREL/AB,BI
L2 0 S TUMOR NECROSIS FACTOR FAMIL Y RELATED
L3 53 S TUMOR NECROSIS FACTOR FAMIL Y/AB,BI
L4 11 S L3 AND RELATED/AB,BI

FILE MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS
ENTERED AT 10:39:33 ON 10
JUL 2000

L5 9 S L1 OR L2
L6 8 DUP REM L5 (1 DUPLICATE REMOVED)
L7 E BROWNING J/AU
L8 264 S E3-E5
L9 E BROWNING JEFFREY/AU
L10 192 S E1-E9
L11 456 S L7 OR L8
L12 74 S L3 AND TUMOR NECROSIS FACTOR/AB,BI
L13 26 S L10 AND FAMIL Y/AB,BI
L14 16 DUP REM L11 (10 DUPLICATES REMOVED)
E CHICHERPOTICHE YVES/AU

=> s l10 and trel/ab,bi

'AB IS NOT A VALID FIELD CODE
L13 1 L10 AND TREL/AB,BI

=> d bib ab

L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2000 ACS

AN 1998:112459 CAPLUS
DN 128:189180
TI construction and therapeutic use of recombinant gene encoding a
tumor **necrosis*** **factor*** -related ligand
or its

receptor
IN Chicheportiche, Yves; ***Browning, Jeffrey L.***
PA Biogen, Inc., USA; Faculty of Medicine of the University of
Geneva;
Chicheportiche, Yves; Browning, Jeffrey L.
SO PCT Int. Appl. 69 pp.
CODEN: PIXXDD2

DT Patent
LA English
FAN/CNT 1
PATENT NO. KIND DATE APPLICATION NO.
DATE

PI WO 9805783 A1 19980212 WO 1997-US13945
19970807
W. AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN,

CU, CZ, DE,

DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP,
KR, KZ,

LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW,
MX, NO, NZ, PL,

PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA,
UG, US,

UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW, GH, KE, LS, MW, SD, UG, ZW, AT, BE, CH, DE,
DK, ES, FI, FR,

GB, GR, IE, IT, LU, MC, NL, PT, SE, BE, BI, CF, CG, CI,
CM, GA,

GN, ML, MR, NE, SN, TD, TG
AU 9738294 A1 19980225 AU 1997-38294 19970807
CN 1232503 A 19991020 CN 1997-198401 19970807
EP 956351 A1 19991117 EP 1997-955334 19970807

R. AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LT, LU, NL, SE,
MC, PT,

IE, SI, LT, LV, FI, RO
BR 9711046 A 20000111 BR 1997-11046 19970807
NO 9900550 A 19990406 NO 1999-550 19990205
PRAI US 1996-PV23541 19960807

US 1996-PV28515 19961018
US 1997-PV40820 19970318
WO 1997-US13945 19970807

AB ***Tumor*** **necrosis*** **factor*** -related
ligand (
****TREL***) a novel member of the ***tumor***
****necrosis***
****factor*** family (TNF), modified ****TREL***, and
pharmaceutical
comps, comprising them. The ****TREL*** protein or its
receptor may
have anti-cancer and/or immunoregulatory applications. Human
cells
transfected with the ****TREL*** gene may be used in gene
therapy to
treat tumors, autoimmune and inflammatory disease or inherited
genetic
disorders. ****TREL*** -specific monoclonal antibodies and
antisense
RNA against ****TREL*** are also claimed. The methodol is
exemplified by treating human adenocarcinoma cells with
****TREL*** or
****TREL*** homologs.

****TREL*** homologs.

****TREL*** homologs.

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****TREL*** homologs.

****TREL*** homologs.

E9 1 CHICHERA GUY/AU

E10 1 CHICHERA M/F/AU

E11 1 CHICHERA MICHAEL F/AU

E12 3 CHICHEREAU CLAIRE/AU

=> s e2-e3

L14 70 ('CHICHERPOTICHE Y'V/AU OR
'CHICHERPOTICHE YVES'V/AU)

=> s l14 and trel/ab,bi

'AB IS NOT A VALID FIELD CODE
L15 3 L14 AND TREL/AB,BI

=> dup rem l15

PROCESSING COMPLETED FOR L15
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)

=> d l - bib ab

YOU HAVE REQUESTED DATA FROM 3 ANSWERS -
CONTINUE? Y(N)?

L16 ANSWER 1 OF 3 INPADOC COPYRIGHT 2000 EPO

LEVEL 1
AN 127892689 INPADOC ED 20000523 EW 200020 UP
20000523 UW 200020
TI LIGANDO RELACIONADO A FATOR DE NECROSE DE
TUMOR.
IN YVES CHICHERPOTICHE; JEFFREY L. BROWNING
INS ***CHICHERPOTICHE YVES***; BROWNING
JEFFREY L.
PA BIOGEN, INC.; BIOGEN, INC.; THE FACULTY OF
MEDICINE OF THE UNIVERSITY OF
GENEVA; THE FACULTY OF MEDICINE OF THE
UNIVERSITY OF GENEVA
PAS BIOGEN INC; FACULTY OF MEDICINE OF THE UNI
PAA US; CH
DT Patent
PIT BRA UNEXAMINED PATENT APPLICATION
PI BR 9711046 A 20000111
AI BR 1997-11046 A 19970807
PRAI US 1996-23541 P 19960807
US 1996-28515 P 19961018
US 1997-40820 P 19970318
WO 1997-US13945 W 19970807
AB Patente de Invenção: LIGANDO RELACIONADO A
FATOR E NECROSE DE
TUMOR<D>. Ligando relacionado a fator de necrose de tumor (
****TREL***) um novo membro da família de fator de necrose
de tumor
(TNF), ****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

****TREL*** modificado, e composto es farmac uticas
compreendendo os mesmos.

L16 ANSWER 2 OF 3 INPADOC COPYRIGHT 2000 EPO
 LEVEL 2
 AN 44303990 INPADOC EW 199923 UW 199926
 TI TUMORNEKROSEFAKTOR-RELATERT LIGAND (***TRELL***), ET NYTT MEDLEM AV TUMORNEKROSEFAKTORFAMILJEN (TNF), MODIFISERT ***TRELL*** OG FARMAS
 YTSKE SAAMENSETNINGER INNEHOLDENDE SLIKE IN CHICHERORTICHE, YVES, BROWNING, JEFFREY L. INS ***CHICHERORTICHE YVES***; BROWNING JEFFREY L.
 INA CH, US
 PA BIOGEN INC
 PAS BIOGEN INC
 PAA US
 DT Patent
 PIT NOA0 APPLICATION FILED
 PI NO 9900530 A0 19990205
 AI NO 1999-530 A 19990205
 PRAI US 1996-23341 P 19960807
 US 1996-28315 P 19961018
 US 1997-40820 P 19970318
 WO 1997-US13945 W 19970807
 L16 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2000 ACS
 AN 1998-112459 CAPLUS
 DN 128:189180
 TI construction and therapeutic use of recombinant gene encoding a tumor
 necrosis factor-related ligand or its receptor
 IN ***Chicheportiche, Yves***; Browning, Jeffrey L.
 PA Biogen, Inc., USA, Faculty of Medicine of the University of Geneva,
 Chicheportiche, Yves; Browning, Jeffrey L.
 SO PCT Int. Appl. 69 pp.
 CODEN: PDXD2
 DT Patent
 LA English
 FANCNT 1
 PATENT NO. KIND DATE APPLICATION NO.
 DATE
 PI WO 9805783 AI 19980212 WO 1997-US13945
 19970807
 W, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
 DK, EE, ES, FI, GB, GE, GR, HU, IL, IS, JP, KE, KG, KP, KR, KZ,
 LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
 PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US,
 UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW, GH, KE, LS, MW, SD, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR,

GB, GR, IE, IT, LU, MC, NL, PT, SE, BE, BI, CF, CG, CL, CM, GA,
 GN, ML, MR, NE, SN, TD, TG
 AU 9738294 AI 19980225 AU 1997-38294 19970807
 CN 1232503 A 19991020 CN 1997-198401 19970807
 EP 956351 AI 19991117 EP 1997-93334 19970807
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, IL, LU, NL, SE, MC, PT,
 IE, SL, LT, LV, FI, RO
 BR 9711046 A 20000111 BR 1997-11046 19970807
 NO 9900530 A 19990406 NO 1999-530 19990205
 PRAI US 1996-PV23341 19960807
 US 1996-PV28315 19961018
 US 1997-PV40820 19970318
 WO 1997-US13945 19970807
 AB Tumor necrosis factor-related ligand (***TRELL***), a novel member of the tumor necrosis factor family (TNF), modified ***TRELL***, and pharmaceutical compns. comprising them. The ***TRELL*** protein or its receptor may have anti-cancer and/or immunoregulatory applications.
 Human cells transfected with the ***TRELL*** gene may be used in gene therapy to treat tumors, autoimmune and inflammatory disease or inherited genetic disorders. ***TRELL***-specific monoclonal antibodies and antisense RNA against ***TRELL*** are also claimed. The method, is exemplified by treating human adenocarcinoma cells with ***TRELL*** or ***TRELL*** homologs.
 => d his
 (FILE HOME ENTERED AT 10:33:47 ON 10 JUL 2000)
 FILE MEDLINE ENTERED AT 10:33:58 ON 10 JUL 2000
 L1 1 S TRELL/AB, BI
 L2 0 S TUMOR NECROSIS FACTOR FAMIL Y RELATED PROTEIN/AB, BI
 L3 53 S TUMOR NECROSIS FACTOR FAMIL Y/AB, BI
 L4 11 S L3 AND RELATED/AB, BI
 FILE MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS ENTERED AT 10:39:33 ON 10 JUL 2000
 L5 9 S L1 OR L2
 L6 8 DUP REM L5 (1 DUPLICATE REMOVED)
 L7 E BROWNING JAU
 L8 264 S E3-E5
 E BROWNING JEFFREY/AV
 192 S E1-E9

L9 456 S L7 OR L8
 L10 74 S L9 AND TUMOR NECROSIS FACTOR/AB, BI
 L11 26 S L10 AND FAMIL Y/AB, BI
 L12 16 DUP REM L11 (10 DUPLICATES REMOVED)
 L13 E CHICHERORTICHE YVES/AV
 1 S L10 AND TRELL/AB, BI
 E CHICHERORTICHE YVES/AV
 L14 70 S E2-E3
 3 S L14 AND TRELL/AB, BI
 L15
 L16 3 DUP REM L15 (1 DUPLICATES REMOVED)
 => s 11
 'AB IS NOT A VALID FIELD CODE
 L17 9 L1
 => dup rem 117
 PROCESSING COMPLETED FOR L17
 L18 8 DUP REM L17 (1 DUPLICATE REMOVED)
 => d 1-bib ab
 YOU HAVE REQUESTED DATA FROM 8 ANSWERS - CONTINUE? Y/(N)?y
 L18 ANSWER 1 OF 8 INPADOC COPYRIGHT 2000 EPO
 LEVEL 1
 AN 127892689 INPADOC ED 20000523 EW 200020 UP 20000523 UW 200020
 TI LIGANDO RELACIONADO A FATOR DE NECROSE DE TUMOR
 IN YVES CHICHERORTICHE, JEFFREY L, BROWNING INS CHICHERORTICHE YVES, BROWNING JEFFREY L PA BIOGEN, INC.; BIOGEN, INC.; THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA, THE FACULTY OF MEDICINE OF THE UNIVERSITY OF GENEVA
 PAS BIOGEN INC; FACULTY OF MEDICINE OF THE UNI PAA US; CH
 DT Patent
 PIT BRA UNEXAMINED PATENT APPLICATION
 PI BR 9711046 A 20000111
 AI BR 1997-11046 A 19970807
 PRAI US 1996-23341 P 19960807
 US 1996-28315 P 19961018
 US 1997-40820 P 19970318
 WO 1997-US13945 W 19970807
 AB Patente de Invenção: LIGANDO RELACIONADO A FATOR E NECROSE DE TUMOR
 TUMOR<>D>. Ligando relacionado a fator de necrose de tumor (***TRELL***), um novo membro da família de fator de necrose de tumor (TNF), ***TRELL*** modificado, e compoies es farmac uticas compreendendo os mesmos.

L18 ANSWER 2 OF 8 INPADOC COPYRIGHT 2000 EPO

LEVEL 2

AN 4430390 INPADOC EIW 199923 UW 199926

TI TUMORNEKROSEFAKTOR-RELATERT LIGAND (

TRELL) ET NYTT MEDLEM AV

TUMORNEKROSEFAKTORFAMILJEN (TNF), MODIFISERT

TRELL OG FARMAS

YTISKE SAMMENSETNINGER INNEHOLDENDE SLIKE

IN CHICHEPORTICHE, YVES, BROWNING, JEFFREY L.

INS CHICHEPORTICHE YVES, BROWNING JEFFREY L

INA CH, US

PA BIOGEN INC

PAS BIOGEN INC

PAA US

DT Patent

PIT NOA0 APPLICATION FILED

PI NO 9900550 A0 19990205

AI NO 1999-550 A 19990205

PRAI US 1996-23541 P 19960807

US 1996-28515 P 19961018

US 1997-40820 P 19970318

WO 1997-US13945 W 19970807

L18 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2000 ACS

AN 1998:112459 CAPLUS

DN 128:189180

TI construction and therapeutic use of recombinant gene encoding a

tumor

necrosis factor-related ligand or its receptor

IN Chideportiche, Yves, Browning, Jeffrey L.

PA Biogen, Inc., USA, Faculty of Medicine of the University of

Geneva,

Chideportiche, Yves, Browning, Jeffrey L.

SO PCT Int. Appl. 69 pp.

CODEN: PDXD2

DT Patent

LA English

FAN/CNT 1

PATENT NO. KIND DATE APPLICATION NO.

DATE

PI WO 9805783 AI 19980212 WO 1997-US13945

19970807

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN,

CU, CZ, DE,

DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP,

KR, KZ,

LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW,

MX, NO, NZ, PL,

PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA,

UG, US,

UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW, GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE,

DK, ES, FI, FR,

GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BI, CF, CG, CI,

CM, GA,

GN, ML, MR, NE, SN, TD, TG

AU 9738294 AI 19980225 AU 1997-38294 19970807

CN 1232503 A 19991020 CN 1997-198401 19970807

EP 956351 AI 19991117 EP 1997-95334 19970807

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, IL, LU, NL, SE,

MC, PT,

IE, SL, LT, LV, FI, RO

BR 9711046 A 20000111 BR 1997-11046 19970807

NO 9900550 A 19990406 NO 1999-550 19990205

PRAI US 1996-PV23541 19960807

US 1996-PV28515 19961018

US 1997-PV40820 19970318

WO 1997-US13945 19970807

AB Tumor necrosis factor-related ligand (***TRELL***), a novel

member of

the tumor necrosis factor family (TNF), modified ***TRELL***

, and

pharmaceutical compns. comprising them. The ***TRELL***

protein or

its receptor may have anti-cancer and/or immunoregulatory

applications.

Human cells transfected with the ***TRELL*** gene may be

used in gene

therapy to treat tumors, autoimmune and inflammatory disease or

inherited

genetic disorders. ***TRELL*** -specific monoclonal

antibodies and

antisense RNA against ***TRELL*** are also claimed. The

method, is

exemplified by treating human adenocarcinoma cells with

TRELL or

TRELL homologs.

L18 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2000 BIOSIS

AN 1997:49723 BIOSIS

DN PREV19979796436

TI Morphoanatomical studies of Furcraea (Agavaceae) of India.

AU Khan, Hafiz Ahmed

CS Birbal Sahni Inst. Palaeobotany, 53 University Rd., Lucknow

226007 India

SO Journal of Plant Anatomy and Morphology (Udipur), (1997)

Vol. 7, No. 2,

pp. 140-147,

ISSN: 0256-436X

DT Article, (TAXONOMIC KEY)

LA English

AB Few species of Furcraea Vent. have been introduced in India as

garden and

hedge plants, and for obtaining fibre. These are succulent plants

like

Agave and are growing in dry, tropical and subtropical places

throughout

the country. F. gigantea Vent. is a common species and a more

important

plant known as Mauritius Hemp. Other species grown in India are

beddinghausii Koeh., F. longeva Karw. & Zucc. F. sellos C. Koeh.

var. marginata ***Trell***, and F. hexapetala Urb. The botanical

identity

of south Indian species known as Mauritius Hemp is F. hexapetala

Urb.

(Syn. F. cubensis Haw.) and not F. gigantea Vent. The F. gigantea

is a

large shrub with fleshy leaves possessing a brown tip spine and

armed or

often Basal part only, armed margins. Trunk is long below the

rosette of

leaves. A variety of F. gigantea is mediotpecta which is variegated

with

butter coloured strips along the leaves. This variety is generally

grown

as ornamental in the gardens in pots or on the ground. Leaves of

willemetiana, the other variety are light green coloured, armed with

prickles and the juice is of mild odour. Variety marginata of F.

sellos

has the leaf margins armed with distant brown horny hooked

prickles.

L18 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2000 ACS

AN 1991:141403 CAPLUS

DN 114:141403

TI Meningococcal class I outer-membrane protein vaccine

IN Seid, Robert C., Jr.; Paradiso, Peter R.; Poolman, Jan T.;

Hoogheutout,

Peter; Wieritz, Emmanuel J. H. J.; Van der Ley, Peter; Heckels,

John

Edward; Clarke, Ian Nicholas

PA Praxis Biologics, Inc., USA, Rijksinstituut voor Volksgezondheid

en

Milliehygiene

SO PCT Int. Appl., 121 pp.

CODEN: PDXD2

DT Patent

LA English

FAN/CNT 1

PATENT NO. KIND DATE APPLICATION NO.

DATE

PI WO 9006696 A2 19900628 WO 1989-US5678

19891219

WO 9006696 A3 19900712

W: AU, DK, FI, JP, NO, US

RW: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE

NL 8803111 A 19900716 NL 1988-3111 19881219

NL 8900036 A 19900716 NL 1989-36 19890106

NL 8901612 A 19900716 NL 1989-1612 19890626

AU 9048219 A1 19900710 AU 1990-48219 19891219

AU 640118 B2 19930819 EP 1990-501397 19891219

EP 449958 A1 19911009 EP 1990-501397 19891219

EP 449958 B1 19950322

R: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE

JP 06503465 T2 19940421 JP 1990-501662 19891219

AT 120093 E 19950415 AT 1990-901397 19891219
ES 2070312 T3 19950601 ES 1990-901397 19891219
CA 2007248 AA 19900706 CA 1990-2007248 19900105
NO 9102369 A 19910806 NO 1991-2369 19910618
DK 9101174 A 19910815 DK 1991-1174 19910618
PRAJ NL 1988-3111 19881219
NL 1989-30 19890106
NL 1989-1612 19890626
NL 1989-36 19890106
WO 1989-US5678 19891219
AB Outer-membrane vesicles, class 1 outer-membrane proteins (OMPs) of
Neisseria meningitidis, fragments or oligopeptide conig. epitopes of
the class 1 OMPs, and antigenic conjugates are provided for
immunization
against meningococcal disease. Also provided are cloning and
prodn. of
fusion proteins conig. class 1 OMP epitopes and flagellin protein.
Epitope sequences are identified, and DNA sequencing of class 1
OMP genes
from different N. Meningitidis serosubtypes is presented. Thus,
recombinant flagellins conig. either a VR1 (1st variable region of
class 1
ONMP), VR2, or a cassette of both VR1 and VR2 are effective in
eliciting
antibody response which was cross-reactive to purified P1.16 (class
1 ONMP
subtype) and, to a lesser extent, to outer-membrane complex. Each
construction also induced significant anti-flagellin titers, control
wild
type flagellin only induced antibody response to flagellin itself.
Recombinant flagellin-oligosaccharide conjugate also prepnd. and
tested.

L18 ANSWER 6 OF 8 EMBASE COPYRIGHT 2000 ELSEVIER
SCI. B. V.
AN 85035271 EMBASE
DN 1985035271
TI The hypertensive genotype.
AU Harald B.
CS Odense University Hospital, Dept. Intern. Med. C, DK-5000
Odense, Denmark
SO Scandinavian Journal of Primary Health Care, (1984) 2/3 (96-97),
CODEN: SJPCD7
CY Sweden
DT Journal
FS 022 Human Genetics
017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
006 Internal Medicine
LA English
AB ***Trell*** and collaborators have tried to define the
hypertensive
genotype by an analysis of risk factors in hypertensive patients with
varying degree of genetic predisposition. The data support the view

that
the genetic predisposition for hypertension is not per se associated
with
such accepted cardiovascular risk factors in the population as high
serum
cholesterol and triglyceride content, and impaired glucose
tolerance. What
is this polygenic predisposition to hypertension like? Gradually it
has
proved possible to define some contributing factors. Increased
sensitivity
to sodium loading - the mechanism known to be active in some
strains of
rats - may result in hypertension in humans as well. An elevated
intracellular sodium concentration with increased smooth muscle
reactiveness has been demonstrated in hypertensive patients. Data
are in
existence supporting a correlation between hypertension and a
number of
varying traits: Certain HLA-alleles, the C3F-allele in the
complement
system, different autoantibodies, herpesvirus antibodies, increased
adrenal responsiveness to angiotensin-II, increased catecholamine
release
during exercise, a high proportion of fast twitch fibres in skeletal
muscles. Probably this spectrum of characteristics will be further
broadened in the future. The genetic predisposition to hypertension
must
be considered the result of the presence or absence of these traits.
The
person who, at the same time, is salt sensitive, C3F positive, with a
high
proportion of fast twitch muscle fibres, etc is particularly
predisposed.
Today it is not possible to single out the relative importance of
individual factors in the pathogenesis of human hypertension. Nor
can we
predict to what extent a diagnostic disentanglement along these
lines
should determine the therapeutic strategy.

L18 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1982:174918 BIOSIS
DN BA73:34902
TI HIRSUTINOLIDES FROM VERNONIA-SP.
AU BOHLMANN F, MUELLER L, GUPTA R K, KING R M,
ROBINSON H
CS INST. ORG. CHEM., TECHNICAL UNIV. BERLIN, D-1000
BERLIN 12, W. GER.
SO PHYTOCHEMISTRY (OXY), (1981) 20 (9), 2233-2238.
CODEN: PYTCAS, ISSN: 0031-9422.
FS BA, OLD
LA English
AB Of the 19 spp. of Vernonia [V. alameda H. Robins., V.
condensata Baker, V.
corticea Less, V. echinifolia Mart., V. farinosa Baker, V. gigantea
Trell, Branner et Cor., V. hagei H. Robins., V.

holosericea Mart.
ex DC, V. intermedia DC, V. kunzei Hieron., V. laxa Gardn., V.
mariana
Mart., V. missionis Gardn., V. myrsinitis Ekman, V. obtusata Less,
V.
regis H. Robins., V. texeirens H. Robins., V. tomentella Mart. and
V.
venosissima Sch. Bip. ex Baker] studied, 5 contained highly
oxygenated
sesquiterpene lactones, the rest contained predominantly
triterpenes,
especially lupane derivatives.

L18 ANSWER 8 OF 8 MEDLINE DUPLICATE 1
AN 76058643 MEDLINE
DN 76058643
TI Hydatonin derivatives and malignancies of the haemopoietic
system.
AU Bichel J
SO ACTA MEDICA SCANDINAVICA, (1975 Oct) 198 (4) 327-8.
Journal code: 14G, ISSN: 0001-6101.
CY Sweden
DT Journal, Article, (JOURNAL ARTICLE)
LA English
FS Abtitled Index Medicus Journals, Priority Journals
EM 197603
AB Two patients are described who developed malignant lymphoma
(lymphosarcoma) after diphtherydantoin therapy because of
epilepsy.
Malignant lymphoma in a few patients receiving this medication
has been
described earlier. The literature has been reviewed and discussed
recently
by Rausing and ***Trell*** (2).

=> file stringide

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FULL ESTIMATED COST	128.86	133.99	
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)			
SINCE FILE TOTAL			
CA SUBSCRIBER PRICE	ENTRY	SESSION	
	-5.01	-5.01	

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JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND
FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 7, 2000 (20000707LUP),

=> file medline

COST IN U.S. DOLLARS	ENTRY	SINCE FILE	TOTAL
FULL ESTIMATED COST	SESSION	0.00	133.99
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)			
SINCE FILE	TOTAL	ENTRY	SESSION
CA SUBSCRIBER PRICE		0.00	-5.01

FILE MEDLINE ENTERED AT 10:56:01 ON 10 JUL 2000
FILE LAST UPDATED: 6 JUL 2000 (20000706UP). FILE
COVERS 1960 TO DATE.

MEDLINE has been reloaded to reflect the annual MeSH changes
made by
the National Library of Medicine for 2000. Enter HELP RLOAD for
details.

OLDMEDLINE, data from 1960 through 1965 from the Cumulated
Index
Medicus (CIMA), has been added to MEDLINE. See HELP
CONTENT for details.

Left, right, and simultaneous left and right truncation are available in
the
Basic Index. See HELP SFIELDS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY
AND ACCURATE
SUBSTANCE IDENTIFICATION.

=> d his

(FILE HOME ENTERED AT 10:33:47 ON 10 JUL 2000)

FILE MEDLINE ENTERED AT 10:33:58 ON 10 JUL 2000
L1 1 S TRELL/AB,BI
L2 0 S TUMOR NECROSIS FACTOR FAMILY RELATED
PROTEIN#/AB,BI
L3 53 S TUMOR NECROSIS FACTOR FAMILY/AB,BI
L4 11 S L3 AND RELATED/AB,BI

FILE MEDLINE, EMBASE, BIOSIS, INPADOC, CAPLUS
ENTERED AT 10:39:33 ON 10
JUL 2000

L5 9 S L1 OR L2
L6 8 DUP REM L5 (1 DUPLICATE REMOVED)
E BROWNING J/AU
L7 264 S E3-E5
E BROWNING JEFFREY/AU
L8 192 S E1-E9
L9 456 S L7 OR L8
L10 74 S L9 AND TUMOR NECROSIS FACTOR/AB,BI

L11 26 S L10 AND FAMILY/AB,BI
L12 16 DUP REM L11 (10 DUPLICATES REMOVED)
E CHICHPORTICHE YVES/AU

L13 1 S L10 AND TRELL/AB,BI
E CHICHPORTICHE YVES/AU
L14 70 S E2-E3

L15 3 S L14 AND TRELL/AB,BI
L16 3 DUP REM L15 (0 DUPLICATES REMOVED)
L17 9 S L1
L18 8 DUP REM L17 (1 DUPLICATE REMOVED)

FILE STNGUIDE ENTERED AT 10:52:27 ON 10 JUL 2000

FILE MEDLINE ENTERED AT 10:56:01 ON 10 JUL 2000

=>

--Logging off of STN--

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	ENTRY	SINCE FILE	TOTAL
FULL ESTIMATED COST	SESSION	0.30	134.29
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)			
SINCE FILE	TOTAL	ENTRY	SESSION
CA SUBSCRIBER PRICE		0.00	-5.01

STN INTERNATIONAL LOGOFF AT 10:56:18 ON 10 JUL 2000